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# Observations on Exudative Epidermitis

LLOYD D. JONES, D.V.M., M.S.
South Dakota State College
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"Greasy pig disease" is the common term for this condition. The etiology is not known to date, but in this paper Dr. Jones presents the possible causes and evidence to support or refute each. He also reviews experimental work that has been conducted and factors to consider in differential diagnosis.

His work, published in 1956, is the only domestic paper that has been supported by any investigation on this condition.

Exudative epidermitis is in all cases a generalized dermatitis involving the entire body surface of piglets. It is characterized by sudden onset and short course. There is hyperhidrosis, excess sebaceous secretion, exfoliation, exudation, no pruritus and loss of skin function. Extreme dehydration occurs and rapid exhaustion, usually terminating in death. A secondary bacterial invasion accompanies the process.[22] It follows a progressive course, which may be divided on a time basis into peracute, acute or subacute forms.

This is not a new disease. It was described first by Spinola in 1842 in Germany. Since then, reports of its appearance have been made from other European countries, the British Isles, Japan, Australia and the United States. It was probably first recognized in North America in 1911.[25] Since then, its occurrence has been reported by numerous workers. Except for brief considerations at veterinary association meetings, little attention has been paid to this disease for the last 25 years. It was assumed to be 1 of the causes of baby pig losses to be accepted as such.

The disease is widespread throughout the middle west. It was thought to be found only in areas where the pig population was dense; however, its appearance has been reported from less densely populated areas. Its prevalence has increased during the past several years. The disease attacks young pig at any time between 3 and 35 days of age. Both the morbidity and mortality rates vary greatly. Of those pigs affected the mortality rate is very high.

The nickname of "greasy pig" has been most generally assigned to this condition. It is unfortunate that veterinary medicine possesses the stigma of ownership to a long list of boorish terminology. Exudative epidermitis was chosen after basic studies of the disease were completed. Exudative epidermitis is as all-inclusive in definition as knowledge permits to date, from its genesis to the etiology. When the cause is known, it is hoped we can justify a name that will dignify our profession, as has been suggested by Seaton.[27]

*These are typical skin lesions of exudative epidermitis. The skin is very wet and sticky.*

## Possible Causes

No specific predisposing causal factors have been found to date. There are several factors which appear appropriate at this time for consideration in search for the specific cause of exudative epidermitis.

New drugs, antibiotics, radiation and agricultural chemicals are the hottest numbers on the market today. All of these may be ruled out. Exudative epidermitis appeared 100 years earlier. Any increased incidence can probably be accounted for by an increased alertness, an awareness of the disease, an increase in pig populations and more concentrated husbandry practices. It should be noted that husbandry practices have greatly improved since the days when poor management and lack of sanitation were accused of being the cause.

*Nutritional:* Hogan reported the appearance of a generalized dermatitis in some 30-day-old pigs which were being used for nutritional studies.[17] Pictures of his pigs illustrate some lesions that are quite suggestive. In another report concerning some nutritional experiments there are 3 more illustrations of pigs afflicted with an equally suggestive dermatitis.[18]

A generalized dermatitis was produced in some young pigs being maitained on an experimental avitaminosis-A diet. In addition to the dermatitis, these pigs exhibited symptoms of a progressive disturbance involving the central nervous system.[15]

Dermatitis and other lesions have been produced in pigs kept on a fat-deficient diet.[45] Hanson believes that both parakeratosis of swine and exudative epidermitis are 2 different stages or phases of the same disease, due to a deficiency of dietary essential fatty acids.[14] There is little confirming evidence available to support this view.

There are strong indications that pyridoxine is required by pigs for normal skin growth just as it is required for man, rats and dogs.[42] Extensive studies were made of 23 human patients with seborrheic dermatitis.[30] It was concluded from these studies that pyridoxine is metabolically active or can be converted to a metabolically active substance in the skin. These studies suggest

that patients with seborrheic dermatitis have a metabolic defect in the skin. Furthermore, they present a concept of disease due to a local deficiency state, possibly occurring because of an inborn or acquired metabolic error, which greatly increases the requirement for an essential nutrient.

To better appreciate the application of human medicine, it should be interjected here that the skin of the pig is very similar in most respects to the skin of man. The only real differences are those of thickness and the disposition of the hair.

Some experiments conducted using young white pigs maintained on a pellagra-producing diet produced a generalized dermatitis along with severe gastro-intestinal disturbances.[7] Recovery followed the use of nicotinic acid.

A ration composed of yellow corn, low protein tankage, alfalfa and salt was inadequate for weanling pigs weighing 30 pounds. The lesions produced included rough hair coats and a dermatitis suggestive of this disease.[29]

Studies were made of 80 cases of nutritional enteritis in pigs. These studies indicate that deficiencies of niacin, pantothenic acid, and possibly riboflavin, were involved in producing symptoms of the disease.[28] The report includes a picture of a group of pigs clearly showing evidence of a generalized dermatitis. These observations are supported by similar reports of other investigations.[19,20,21,31] A generalized dermatitis, along with other lesions, has been produced in a group of experimental pigs maintained on a riboflavin-restricted diet.[30] From a study of these and many other reports it becomes plainly obvious how essential a role the B-vitamins play in the health and well-being of the pig. It is generally recognized that nutritional diseases may be reflected through 2 or even 3 generations.

*Anemia:* This subject is brought up every time diseases of baby pigs are discussed. Nutritional anemia was first regarded as due to a simple iron deficiency. Later the association of cooper to iron metabolism was demonstrated; and now cobalt

*This photograph shows well developed skin lesions of the early phase of the disease.*

has been added to the essential list as a catalyst.

A pyridoxine deficiency produces an anemia in swine. There is an increase in the rate of iron migration from the serum into the body tissues. Thus, even though abundant amounts of total body iron are present, it is not available for the production of erythrocyte hemoglobin.[8] Nor will supplemental pyridoxine furnished alone completely relieve the anemia once it is established.

In reports on anemia by Doyle[10] and Hamilton [18] are shown illustrations of pigs with a dermatitis which might be assumed to be an antecedent for exudative epidermitis. A certain amount of controversy and confusion apparently exists concerning the matter of anemia in pigs. The subject apparently is in real need of additional studies and revaluation.

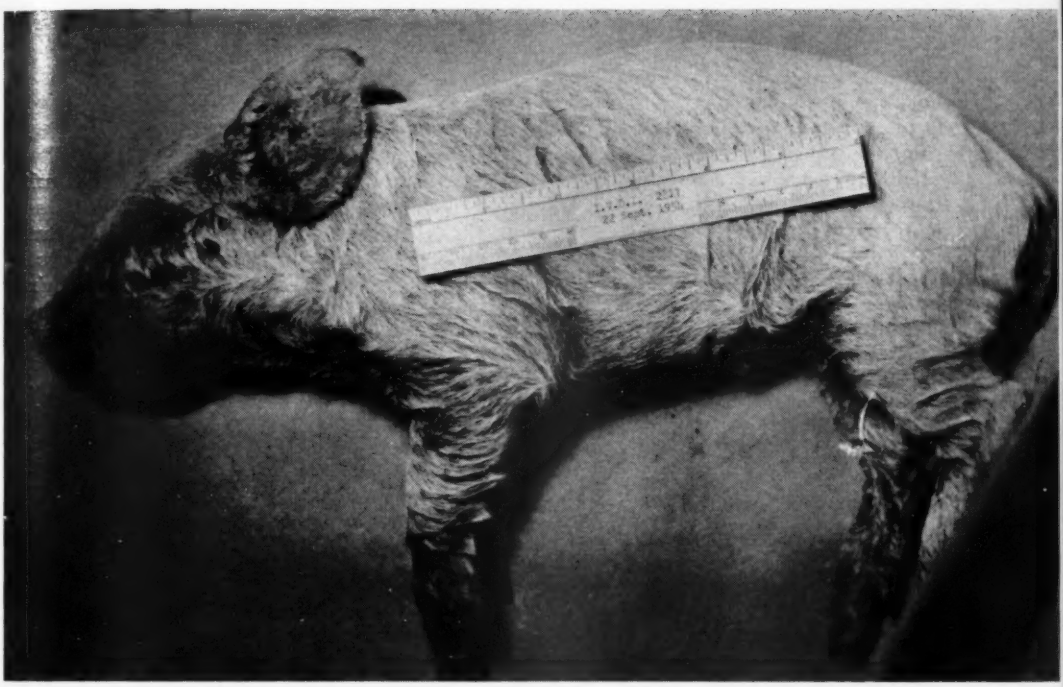
Nutritional anemia should not be confused with the so-called "normal" or "physiological" form of anemia of baby pigs. Starting on the day following birth there is a gradual fall in hemoglobin level, total leucocyte count and erythrocyte numbers. This fall in these blood values occurs gradually and continues until the 7th to the 15th day, after which period these values gradually increase to reach normal values about the 21st day.[9,24,33] This type of anemia has not been credited with disease predisposition.[43] Under these conditions undue credit may be assigned to the effects of administering supplemental iron when actually it is contraindicated. Thus, here are cited 2 conditions offering possibilities of serious hazards connected with toxicity from iron therapy when used in cases where it is not required.

A study of the blood picture associated with exudative epidermitis presents a most difficult interpretation. The problem is confounded with the so-called "physiological" anemia with an associated hemoconcentration, plus a hemoconcentration resulting from the rapid dehydration associated with the disease.

*Metabolic derangements:* Postmaturity of the human infant has been defined as the child 300 days or more in gestation. In this disease the vernix disappears, the exposed skin of the fetus desquamates and is macerated. After delivery the child's skin is dry, cracked, tends to peel and is stained or pigmented. The cause has variously been attributed to placental dysfunction, hormonal imbalance and intangible nutritional disturbances.[8] The condition has been experimentally produced in doe rabbits by administering hormones. Prolonged gestation has been reported in cattle.[44]

It is a very difficult problem to ascertain the exact time of conception in the multiparous sow. In studies of variations in farrowing it was found that the length of the gestation period showed a rather wide range from 98 to 124 days. It was also found that there was a slight tendency for some of the litters which were carried longer than the average to be smaller than those which were farrowed earlier than the average.[5] It has been pointed out that the larger litters are made up by smaller individuals with less maturity at the time of birth.[34] Prolonged gestation was produced in a group of second generation gilts maintained on a complete type ration in which all protein was of plant origin. In addition there were prenatal deaths in some cases.[42]

*Allergy:* The role of allergy as a possible etiological factor remains yet to be investigated. Eosinophilia is one of the many consistent and outstanding features of exudative epidermitis. It appears early and increases as the disease progresses. Infiltrative eosinophilia is one of the many extrarenal lesions of uremia, which is another consistent lesion associated with exudative epidermitis. Eosinophilia has for a long time been associated with allergy and as a result of an antigen-antibody reaction in tissues. The antigen-antibody reaction phenomenon includes certain cases of dermatitis, blood dyscrasias, brain damage and collagen diseases. These result from the action of autoantibodies developed against tissues made autoantigenic by some alteration to



*A moribund pig approximately 16 days old showing the acute form of the disease.*

the tissue cells.[19] Such an alteration may be caused by infections, drugs and other chemicals, and by physical agents including heat and cold. Periarteritis nodosa is classified within this category. Closely related to this group might also be included the isohemolytic diseases of newborn pigs, marked by anemia, icterus and death following the intake of interfering colostral antibody.

Cellular pathology of exudative epidermitis skin will reveal a hyperplasia and hypertrophy of the stratum spinosum with both inter- and intracellular edema. These lesions are generalized and there are no signs of pruritus. This microscopic picture is very different from the one produced by injury or irritation to the skin. Injury produces a thickening of the stratum corneum that in turn plugs or pinches the openings of the sweat pores. Then sweat is secreted, not out onto the skin but instead, into the skin, producing marked inflammation and itching. This type of a reaction is not generalized but focal and involves only the area of damage. Exudative epidermitis always presents a generalized reactive skin. Microscopically it is characterized by marked activity of the stratum cylindricum, and marked activity and thickening of the stratum spinosum.

*Bacteriological:* A study of the reports on exudative epidermitis was made by Pillsbury.[20] He believes that they certainly strongly suggest some infectious process, even in the absence of demonstrating bacteria which seem to be pathogenic and without evidence of inflammatory reaction. In the studies he conducted on newborn laboratory animals, infection was produced with ease by strains of staphylococci which

would not produce infection in older animals. These infectious processes progressed with great speed. They are likened to impetigo in human newborns, where there is often comparatively little inflammatory response along with an occasional overwhelming toxemia. The similarity of the skin of man and pig is at its nearest approximation when the pig is young.

Several attempts have been made to transmit the disease in young pigs with cultures of staphylococci.[39] A focal dermatitis was produced at the site of inoculation but spreading did not occur. The local effect of staphylococcus alpha toxin has been studied.[4,41] The toxin acted directly on the arteries and veins, causing vasospasm, vasoconstriction, blanching of the tissues and necrosis. Exudative epidermitis is notably marked by a severe erythema. The microorganisms usually found associated with the disease have been identified as *Staphylococcus epidermidis*, which has non-pathogenic characteristics. This does not settle the problem however. The pathogenicity of staphylococcus has been determined indirectly by the use of coagulase. Recently, it has been demonstrated that coagulase-negative strains of staphylococci may be turned into pathogenic coagulase-positive forms.[11] Staphylococci introduced into normal tissue tend to remain localized.[28] Virulence and invasiveness connote 2 different properties of bacteria and viruses. Tellurite reduction has now been proposed as a more efficient indirect indicator of potentially pathogenic staphylococci.[16]

In studies on the specificity and nature of the phenomenon of local skin reactivity to various bacterial filtrates, it has been demonstrated that the skin-preparatory and reacting factors of various unrelated microorganisms are able to substitute for each other, provided they have the power of eliciting the phenomenon for themselves. The list of such capable organisms also includes certain tumor extracts and epitheliotropic viruses as vaccinia.[1] These relationships leave open ample opportunity for further study. Here again a so-called "orphan" virus may find its heritage and *Staph. epidermidis* a partner.

## Differential Diagnosis

A milder form of exudative epidermitis has been reported appearing in Kansas.[29] The pigs were 5 to 7 weeks old, weaned and fast-growing. On the ventral parts of their body were circular reddened ringworm-like lesions. In 1 clinical case, rapid and complete recovery followed the addition to the ration of an ounce of corn or soybean oil per pig for 10 days. Improvement occurred in many pigs without treatment, but more slowly. This type of the disease may be accounted for with another case reported involving an enteritis with dermatitis.[2] The

*The skin of the entire body, including that of the ears, nose, feet and tail, is always involved.*

*Typical feet lesions found in the acute form.*

disease occurred mostly in large litters about 3 weeks old. Usually all were affected, emaciated, afebrile, had a yellowish-white diarrhea, a conjunctivitis and exudative dermatitis with brown crusts on the face and later on other parts of the body. The mortality was low and no bacteria were cultured. The cause of the condition was attributed to nutritional inadequacy due to depletion of sow's milk. Recovery followed witholding solid food from the piglets and supplementing their diets with milk, plus supplementing the sow's diet and getting both out onto grass pasture. Both of these cases suggest they might be similar to those described as erythema multiforme or pityriasis rosea.

A case of exudative epidermitis was encountered in Australia.[5] A 4-week-old pig had been ill 1 week before death, 4 of the 14 in the litter had died at birth and another at 3 weeks of age. Microscopic degenerative changes were found in the cerebral cortex, the lenticular nucleus and Purkinje cells of the cerebellum. This is the first case reporting symptoms and lesions involving the nervous system. Lice were present on the pigs, and the sow's feed was very moldy. Since the report of this case, Luke has examined the brains of several Irish pigs from as many different cases of exudative epidermitis with negative results.[27] A microscopic congenital cerebellar anomaly in pigs has been reported.[28] These pigs were unthrifty, showed ataxia, dizziness, incoordination and all died. Microscopic examinations of brain material revealed misshapen and malformed nuclei of the Purkinje cells in the cerebellum.

## Experimental Work

Transmission experiments with exudative epidermitis were conducted by Klobuk.[28] He found it to be a specific infectious disease, easily transmitted artifically, with recovery resulting in acquired immunity. He successfully transmitted the infection by inoculations with a filtrate prepared from an emulsion of the skin of affected animals. He decided that the causal agent "probably is a virus." Descriptions and other details of his work are not sufficiently clear. He stated that re-examination of his work is desirable.

Some transmission experiments were conducted using white pigs that were 7

days of age. These pigs were obtained from an isolated farm with low incidence of swine diseases, including hog cholera and erysipelas. On the day of procurement they were transported in clean equipment to the laboratory and placed in isolation. Each pig was given homologous anti-hog cholera serum and then challenged by scarification in the axillary region with a liberal amount of material prepared from lymph node and skin material from 4 different field cases of exudative epidermitis. The pigs were maintained on a synthetic diet including powdered whole milk from cows. Temperatures and clinical observations were made for 21 days. A light illness and a mild dermatitis developed over the neck, shoulders and withers of 2 pigs. The skin was hyperesthetic, thickened and scurfy. The condition lasted several days but cleared up shortly. On the 21st day necropsies revealed the presence of no gross lesions and bacteriological studies were negative.

The results of this experiment seriously warrant continuation and further studies along these lines but under more critical conditions. This would include the use of even younger susceptible pigs that are disease-free and devoid of antibody from colostral, immunological and dietary sources.

## Treatment

Specific therapy cannot be stated. No medication has been found to be of value. Any possible change in management should be introduced with a hope of improvement, such as change in pasture, bedding, and yards. Affected litters should be isolated. Other litters should be spread out and segregated as much as possible.

The affected pigs greatly need fluid electrolytes and nutrition. Any local treatment must be most bland, as soaking in warm mineral-free water, followed by bland oils and guarding against loss of body heat.

A variety of treatments have been credited as giving good results, but none have been supported by controlled experiments.

## Summary

Exudative epidermitis is a generalized dermatitis of piglets that is characterized by sudden onset and short course usually terminating in death. There is hyperhidrosis, excessive sebaceous secretion, exfoliation, exudation and loss of skin function. The disease apparently has been present for many years but its prevalence has increased during the past several years. It is found wherever pigs are raised.

No specific predisposing factors or etiological agent have been found to date. Nutritional deficiencies have been considered the cause by some. Dermatitis in young pigs can be produced by vitamin A and fatty acid deficiencies. Deficiencies of B vitamins, particularly pyridoxine, produce skin lesions similar to this condition. Other possibilities of the cause are anemia, metabolic derangements, allergy and staphylococcal infections.

At the present time the only treatment is symptomatic. Lost fluids should be replaced, nutrients provided, skin protected and piglets kept warm. Healthy pigs should be separated from diseased ones.

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*Interior view of a nursery house at Trimble Manor, Trimble, Mo. The pigs are moved here from the farrowing house when they weigh about 14 lb. One litter is placed in each pen. When they average about 40 lb., they are moved from the nursery to a feeding floor.*

## Problems of Raising Swine In Confinement

*Lameness in Sows*

Some of the most difficult problems encountered in confinement swine raising projects are the simple ones that are easily overlooked.

The foot troubles of the heavy sow or pregnant gilt are examples. As the lameness develops, it is difficult to find a cause. Then it is noted that the operator has just built the unit. The concrete floor is rough like sawdust. While he is washing down the floor, these sows come out and try to drink out of the hose. Their feet become wet, and the corium of the hoof softens. When the sow gets up, her front feet slide out in front of her and a layer of hoof is left on the rough concrete floor. It takes only about 2 or 3 days before large swellings develop on 1 of the claws. The metacarpus becomes involved, and the swelling

may even extend up to the knee. A common streptococcus organism is the cause of this infection.

A veterinarian generally sees this condition about 10 to 12 days after it begins. The enlarged, swollen joint is easily confused with erysipelas. These infected animals may even develop a septicemia and die.

*Hygromas*

Concrete hygromas are seen in sows and boars when the concrete floors are a little too smooth. Many operators are building the floors with a slope of 1 inch per foot. This is quite steep for a heavy sow if the surface is too smooth. If the pregnant sow slips a few times, the result may be weak pigs at birth or it may even cause an abortion.

*Vulva Biting*

This is another confinement raising problem. There is much about swine psychology that we do not understand. Why does 1 sow in a pen have to be the "goat"? Frequently, 20 to 30 sows will start working on 1 individual. She gets down, and they will soon eat her. The cause of such problems is not understood.

Vulva biting is exceptionally bad at feeding time when there is not enough feeding space. This is at least one of the causes.

*Over-feeding*

Over-feeding should be avoided. We believe that sows kept in confinement units should gain about 4 lb. per day during the first 90 days of the gestation period. These sows look thin and are constantly squealing for feed.

For the last 25 days of gestation they are put on a nearly full feed. We have found that putting these sows on a flushing ration during the last 1/3 of the gestation period when the fetuses are developing rapidly, causes them to produce large 4½ lb. pigs from gilts.

Over-feeding sows on pasture often contributes to poor milk production. Although it is not always true, the fattest sow is often the poorest milker. Also, there are more farrowing troubles in over-weight sows.

*Farrowing*

The short sows that were raised a few years back had considerable farrowing trouble. Today, less trouble is encountered with the longer, leaner sows being bred. In 2 years experience with over 2,000 sows farrowing, I have performed only 1 cesarean on a sow. That sow had a broken pelvis. In most cases, all that is needed is some pituitrin to stimulate the uterus. Likewise, a retained placenta is rare unless there is an infection.

*Harvey Helms, D.V.M.*
*Trimble Manor*
*Nebraska State Meeting, 1960*

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## Swine Disease Notes

*Notes from a Question and Answer Session on Swine Diseases at the 1960 Nebraska State Meeting.*

*Anaphylactic Reactions in Swine*

An unusually high incidence of anaphylactic reactions in swine has been encountered this past year. The sows were vaccinated with an erysipelas bacterin. After injecting the young pigs at 2 to 3 weeks of age with 1 cc. to 3 cc. of swine anti-erysipelas serum, as many as 50% died. They died so rapidly that adrenalin did not have time to act.

*Case Reports*: 1. The sows had been vaccinated 4 weeks before farrowing. The young pigs were 3 to 4 weeks old and were being given anti-erysipelas serum. By the time 10 pigs had been injected, the first pig was dead. The other 9 pigs were dead in a short time.

2. A group of sick sows were being treated for erysipelas. Each was given 40 cc. of anti-erysipelas serum. Before leaving the farm, one sow was very sick and in a short while 5 more were sick. These pigs

had been vaccinated with an erysipelas bacterin at least 6 weeks previously.

*Erysipelas Treatment*

Large doses of anti-erysipelas serum and antibiotics are more effective in treating erysipelas than antibiotics alone. A sow is given 80 cc. to 100 cc. of serum and 10 cc. of penicillin if she is nursing her pigs. She is not as likely to stop milking following this treatment.

*Erysipelas in Young Pigs*

On some farms it is necessary to treat young pigs before they are 10 days old. In such cases the farmer often administers the serum as soon as they are born. They give a total of 3 cc. in several locations to avoid leakage. It is better to wait until the young pigs are at least 3 days old if possible. These cases are erysipelas. The organisms have been identified on culture.

*Incidence of Navel Ill*

Erysipelas serum, as well as hog cholera serum, reduces the incidence of navel ill. A foreign protein is excellent therapy in this condition. Powered, defatted, sterile milk is good therapy. A dose of ¼ cc. is given. There is a danger of anaphylactic reactions.

*Navel Infections*

Bacterins have been used to control mastitis, uterine infections and navel infections in baby pigs. There is a danger of anaphylactic reactions in the sows if a series of vaccinations are given before farrowing. Mature sows have been killed after the 3rd vaccination.

A 5 cc. dose of mixed porcine bacterin No. 2 given about 15 days before farrowing reduces joint and navel infection by about 12%.

The best method to eliminate navel infections is to catch the young pigs in a sterile bag and to tie the umbilical cord. Staphylococci and streptococci are the organisms that cause the navel infections. The same organisms can be isolated from cases of "bull nose."

## Feeder Pig Problems On Concrete

Feeder pig problems have been accentuated with the advent of concrete pig parlors. Many new problems have been introduced. A higher degree of sanitation, increased knowledge of nutrition, along with new concepts in building, have not solved the problems for the feeder who has a great number of hogs on small areas of concrete.

In any concrete feeding setup every animal that dies should be necropsied. Cultures and sensitivity patterns should be run where necessary. Routine red blood counts, hemoglobin determinations, and fecal examinations should be made. Herd health files, organism culture files, plus a knowledge of local management problems and disease virulence peculiarities of that area, help the local veterinarian to render a valuable service to the feeder.

*Increased Virulence*

Increased virulence of common organisms is one problem that can become serious. The most common instance is erysipelas. A modified erysipelas vaccine provides good immunity in our area, but on concrete, serum and virulent cultures are often necessary to prevent outbreaks.

Apparently, new varieties of organisms appear. For instance, the enterococci which normally are nonpathogenic have caused serious outbreaks of acute pneumonias.

*Cannabalism*

This is very common under certain conditions in concrete parlors. A few simple rules will prevent losses.

1. Give additional mineral mixture free choice.
2. If a hog starts bleeding from a wound or any other reason, remove him from the pen.
3. Put up a swing tire or other playthings to relieve boredom.
4. Isolate sick animals immediately.
5. Take care in introducing new pigs into a full pen.

*Gut Edema*

This is quite a common problem encountered on concrete. Many of our feeder pigs are brought in from Ozark feeder pig sales and placed on full feed. It appears suddenly and always is accompanied by scanty, hard feces. High temperatures and incoordination are the other principal symptoms.

Take the herd off feed immediately, flush with magnesium sulfate, and follow with sulfathiazole in the drinking water for four to five days. If there are moribund animals on arrival, these are given Gallogen (S. E. Massengill Co.) and an injectable antibiotic. This treatment has reduced losses to a low figure, if the client calls when symptoms are first noted.

*Intestinal Infections*

"Necro" is not as common as in swine raised on pasture. More diarrheas are seen from high soybean or a higer protein percentage in the ration than is noted on pasture. When an infectious diarrhea is encountered that becomes a herd problem, cultures are made with SS agar and treatment is according to the organism found and the sensitivity pattern of that organism.

*Lungworms*

These are consistently found on fecal examination, but results with Dictycide (Fort Dodge Laboratories) have been variable. Lungworms are a definite problem, and there should be further study given this problem as well as ways of using this drug to control them.

*Tailbiting*

Dr. R. A. Spring, of Franklin, Ind., has written an excellent article giving cause and treatment of this condition. However, this writer uses a much simpler method that has worked. Post a man by the pen for 15 to 30 minutes and have him pick out the culprit. Over a period of four years, the removal of one pig per pen has eliminated the tailbiting problem.

*Medication*

Both feed and water routes are used, although water medication is the method of choice in most cases. Disease outbreaks on concrete are generally more explosive than on pasture, and often the acutely ill animals will not eat, but will drink. Since pressure water systems are either buried or placed under the concrete, only medications that are completely soluble should be placed in the water. The most popular system used is a 50 gal. barrel with a jet injector so that the medication can be fed into the pressure water line graduated into any percentage.

*Conclusion*

In a confined feeding operation the practitioner will be the important key to profits for the feeder. He must have a detailed preventive medical program. To be able to do an efficient job of preventive medicine and disease control, he must also understand other problems of confinement feeding. Some of these problems are:

1. Number of square feet per hog.
2. Manure disposal—lagoon versus septic tank.
3. Rations—pelleted versus ground versus grain and supplement free choice.
4. Nutrition—makeup of ration.
5. Fog sprays.

These and many other problems that relate to herd health can be worked out between the practitioner, herd owner and feed man.

*W. L. Baker, Jr., D.V.M.*
*Kennett, Mo.*

# Immunization of Swine Against Hog Cholera with a Bovine Enterovirus

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While there are effective methods for preventing hog cholera, the disease itself has continued to remain something of a mystery. Hog cholera was first recorded in Ohio in 1833. This first outbreak was apparently the beginning of an entirely new disease, and from this beginning it has spread worldwide. The disease is not similar to that of any other animal, and apparently there is only 1 antigenic type of the virus. Despite a vast amount of work directed toward discovering a natural reservoir of hog cholera in animals other than swine, it has remained unknown as to how this virus is able to maintain itself between outbreaks. This subject has been thoroughly reviewed by Schwarte.[1] Although hog cholera virus has been shown to be infective for a number of animals including sheep,[2,3] goats,[3] cattle,[3] racoons,[1] rabbits[4] and man,[5] it has not been possible to produce the clinical disease in these species. For these reasons, it was believed that hog cholera virus was totally unrelated to any other mammalian virus.

The report by Darbyshire[6] that there was a serological relationship between mucosal disease of cattle and hog cholera was received with great interest in our laboratory. Following correspondence to determine whether the mucosal disease of cattle to which he referred was the same as the disease complex known in the United States as the virus diarrhea-mucosal disease complex, it was decided to test some of the strains of this group of viruses which we had available in our laboratory to see if they could provide protection against hog cholera in swine. It was known that these viruses would not produce hog cholera, since about a year previously they had been inoculated into a group of pigs to determine if they would produce any disease in this species before proceeding with experiments on cattle which would be kept in close proximity to swine.

The purpose of this article is to describe a series of tests which further demonstrate a definite relationship between hog cholera and the bovine virus diarrhea-mucosal disease complex, which we believe throws considerable light on the history of hog cholera.

## Experimental Methods

Two strains of virus, the Nebraska virus diarrhea virus and the Nebraska mucosal disease virus, were obtained from Mr. N. R. Underdahl, Department of Veterinary Science, University of Nebraska. The Oregon virus diarrhea virus was obtained from Dr.

J. F. Gillespie, Veterinary Virus Research Institute, Ithaca, N. Y. These viruses as received were produced on primary explants of embryonic bovine kidney cells and have been passed twice on this same type of cells in our laboratory. The cells have been grown in a culture medium consisting of Hank's salt solution containing 10% of a 5% solution of lactalbumin hydrolysate, and 100 units penicillin and 100 units dihydrostreptomycin per ml. Just before the virus is inoculated, the medium is changed to one containing Earle's salt solution, lactalbumin and antibiotics.

The virulent hog cholera virus used for these experiments was the same virus used for challenging government inspected tests on hog cholera vaccine and was produced under government supervision from swine acutely sick with hog cholera. The pigs used for these experiments were mostly Hampshires obtained from a source which has supplied hog cholera free pigs for all test purposes. The weight of these pigs when used was between 40 lb. and 100 lb.

## Experimental Results

For the first test, 4 unvaccinated pigs were used. One was not treated and served as a susceptibility and virulence-of-challenge control. Each of the other 3 was inoculated with 5 ml. of a fresh culture preparation of 1 of the 3 enteroviruses: Nebraska virus diarrhea virus, Nebraska mucosal disease virus or Oregon virus diarrhea virus. For each animal, body temperature was taken and a white blood cell count was made prior to inoculation. Daily temperatures and WBCs were determined for 10 days post-inoculation. No pronounced changes in temperature, WBC or clinical appearance were evident during the post-inoculation period.

Fourteen days following inoculation each pig was inoculated with 2 ml. of virulent hog cholera virus. Following exposure to virulent hog cholera virus, all pigs except the 1 inoculated with Oregon virus diarrhea virus succumbed. The pigs that died had symptoms and lesions commonly associated with hog cholera, as determined by temperature, WBC, symptoms, necropsy and bacteriological examination of tissues of dead pigs.

The 2nd test was designed to study further the 1 strain of virus which had protected the pig in the 1st test against virulent hog cholera virus. Sixteen pigs from 2 different farms were used. The animals were stratified by source and randomly assigned to 1 of 3 treatment groups. Ten pigs were each inoculated with 5 ml. of tissue culture grown virus and were designated vaccinates. Three pigs were not inoculated but were penned with the vaccinates to serve as contact controls. The other 3 pigs were not inoculated and were penned separately from the other 13 to serve as isolated controls. Base temperature and WBC was established on each animal before treatment. Daily temperatures and WBCs were taken on vaccinates for 8 days and on contact controls from the 6th to the 8th post-inoculation days.

Fourteen days post-inoculation all 16 pigs were brought together and inoculated with 2 ml. of virulent hog cholera virus. Following challenge the 10 inoculated animals survived while all 6 uninoculated animals, both contact and isolated controls, died or were destroyed *in extremis*. Deaths occurred, as is typical for hog cholera, 7 to 10 days post-challenge, with symptoms and lesions commonly associated with the disease. This test proved that the Oregon virus diarrhea strain protected against hog cholera and that it did not pass from vaccinates to contact controls.

For the 3rd experiment the virus used for inoculation was desiccated from the frozen state under high vacuum. The virus titer of this particular lot was determined by restoring the virus to its original volume with deionized water and measuring the virus content in tissue culture. It had a titer of $10^{-5}$/ml.

Fourteen pigs were randomly assigned into 3 groups of 5, 5 and 4 respectively. Reconstituted virus was inoculated into 10

animals at 2 dosage levels. Five pigs received 2 x $10^4$ virus particles and 5 received 2 x $10^5$ virus particles. The remaining 4 were not treated but were penned with the vaccinates as contact controls.

Twenty-four days post-inoculation each animal was inoculated with 2 ml. of virulent HCV. In addition to the HCV, 1 control pig was inoculated with 60 ml. of anti-hog cholera serum, and 1 other control was injected with a large dose of antibiotics. These 2 pigs served as additional checks on virulence and identity of the challenge virus.

Following challenge the 10 vaccinates and the serum-treated control remained alive and healthy. Both untreated controls and the antibiotic-treated control died with symptoms and lesions commonly associated with hog cholera.

The purpose of the 4th test was to determine if this VD virus would increase in virulence when serially passed in cholera susceptible swine. In 2 parallel series of experiments, blood was serially passaged from 2 virus inoculated pigs to 2 other pigs. Ten milliliters of blood were taken from the "donor" by a sterile syringe and immediately inoculated intramuscularly into the "recipient." Using this procedure, blood passages were made every 7th day.

Twenty to 37 days following inoculation of either the original virus (2 pigs) or passaged blood (10 pigs), each animal was inoculated with 2 ml. of virulent HCV. One additional untreated pig from the same source was also inoculated with HCV. Daily examination during the post-inoculation period revealed no evidence of a virus infection. All pigs remained asymptomatic following inoculation of VD virus or blood from the preceding pig in the respective series. The inoculation and blood passage schedule with post-challenge results are shown in Table 1. Following challenge both virus inoculated pigs, #101 and 201, survived. Pigs #102 and 103 in the 1st series and #202, 203 and 204 in the 2nd series also survived. Pigs #104, 105 and 106 in the 1st series and #205 and 206 in the 2nd, along with the untreated control, died. Symptoms and lesions commonly associated with hog cholera were noted on examination and necropsy of the dead pigs. It was concluded that the virus had been back passaged in swine twice in the 1st series and 3 times in the 2nd series.

It was speculated that the resistance to hog cholera demonstrated by Oregon virus diarrhea virus-inoculated swine might be due to a receptor-blocking mechanism rather than to a humoral or serum antibody.

**Table 1.**

**Schedule of Inoculation, Blood Passage, Exposure to Hog Cholera Virus and Post-Challenge Results.**

| Pig No. | Inoculum | Challenge days post-inoculation | Result |
|---|---|---|---|
| First series. | | | |
| 101 | Desic. VD virus IM | 27 | Survived |
| 102 | 10 ml. blood from 101 IM | 20 | Survived |
| 103 | 10 ml. " " 102 IM | 37 | Survived |
| 104 | 10 ml. " " 103 IM | 30 | Died |
| 105 | 10 ml. " " 104 IM | 23 | Died |
| 106 | 10 ml. " " 105 IM | 20 | Died |
| Second series. | | | |
| 201 | Desic. VD virus IM | 27 | Survived |
| 202 | 10 ml. blood from 201 IM | 20 | Survived |
| 203 | 10 ml. " " 202 IM | 37 | Survived |
| 204 | 10 ml. " " 203 IM | 30 | Survived |
| 205 | 10 ml. " " 204 IM | 23 | Died |
| 206 | 10 ml. " " 205 IM | 20 | Died |
| 04 | Untreated control | | Died |

To determine this point, 2 pigs were hyperimmunized for the preparation of an antiserum by inoculating each with a large dose of tissue culture fluid containing Oregon virus diarrhea virus. Blood was collected from these animals 2, 3 and 4 weeks postinoculation and processed to recover the serum. In a preliminary test, 2 hog cholera susceptible pigs were given doses of this serum, which protected them against challenge with virulent hog cholera virus.

## Discussion

It was reported recently[7] that an antigenic relationship, as determined by serum neutralization tests, exists between the various strains of viruses forming the bovine virus diarrhea-mucosal disease complex. Our first experiment shows that all strains, however, are not sufficiently closely related to hog cholera to provide protection.

Hog cholera is typically a severe disease with mortality approaching 100% in susceptible swine. Virus diarrhea in cattle is generally mild and must usually pass unnoticed since in serological surveys over 50% of the cattle tested had antibodies. Hog cholera virus is believed to represent one serological type while virus diarrhea has quite a wide divergence of serological types.

Zichis[3] showed that hog cholera could be passed into both cattle and sheep by inoculation and then by passing blood from these animals back into swine, he reproduced hog cholera. He also showed that hog cholera virus could be passed to sheep by contact and then back into swine by contact with these apparently normal sheep.

It is entirely possible that mucosal disease virus of cattle could be successfully passed through swine and back into cattle. It is speculated that further antigenic relations between VD and HCV may be demonstrated. Perhaps hog cholera vaccine would be an effective immunizing agent against VD in cattle.

All of this leads to speculation that bovine virus diarrhea represents a virus-host relationship that is long established while hog cholera represents a relationship that has never become compatible. Mitchell[8] suggests that hog cholera virus was a mutant of a virus whose source or original animal host is unknown. Hog cholera could well have developed originally by chance from association of swine with cattle infected with virus diarrhea. If this is indeed the case, it is but an example (the passage of *Leptospira pomona* from swine to cattle is another example) of the danger of keeping these species together.

The fact that it has not been possible to infect pigs kept in contact with other pigs inoculated with Oregon virus diarrhea virus and that it does not back passage very satisfactorily in pigs indicates that this virus is not well adapted to pigs. It would seem that there would be little chance of this particular strain ever transforming itself into hog cholera virus. In contrast to this, hog cholera virus, even after a large number of passages in rabbits, is still capable of changing back into virulent hog cholera if it is serially passed from pig to pig.

The fact that in Canada hog cholera has been eliminated even though cattle and swine are handled much as they are in this country and that there are no regulations concerning movement of cattle because of virus diarrhea or mucosal disease leads to the optimistic viewpoint that if hog cholera could once be eliminated from swine, it could be kept under control.

The immunity produced in swine by this virus has been shown to be related to antibody formation rather than to some type of blocking mechanism. By means of serum tests it is possible to measure the exact amount of antibody produced.

While this series of experiments was started originally to check on the antigenic relationship reported between mucosal disease and hog cholera, increasing interest has developed in the possibility of a useful vaccine. This particular virus has many properties which would make it ideal for

production of a new type of hog cholera vaccine. First of all, it grows in tissue culture upon cells obtained from an animal other than the pig. This eliminates the possibility of contamination by extraneous porcine viruses. Since the virus grows in tissue culture and produces a distinct cytopathic effect, it is possible to make precise measurements of both virus titer and antibody in serum. The virus is extremely mild and is capable of stimulating antibody formation without causing temperature rise or other indication of infection in swine. It apparently does not spread by contact from pig to pig, and it is unlikely that it would become virulent for swine.

Perhaps a vaccine produced from this strain of virus would aid in hog cholera eradication. This might be accomplished by a countywide, statewide and nationwide vaccination program in which every pig was vaccinated for a period of 3 years. Such a program could break the cycle of hog cholera without introducing a virus capable of reverting to virulent hog cholera.


## Summary

One of 3 bovine enteroviruses when inoculated into swine was shown to protect against subsequent exposure to HCV. Following inoculation into swine, no evidence of a virus reaction was observed as measured by WBC, febrile response, reduction in feed consumption or clinical appearance. The virus did not spread from inoculated to uninoculated swine in close contact. The virus can be desiccated and retain its antigenicity. On serial passage in swine the virus did not increase in virulence, but seemed to disappear. Immunity to HC developed in response to inoculation of VD virus is antibody in nature.


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## Atrophic Rhinitis

Some of the thoughts presented by Nils—Erik Bjorklund of the State Veterinary Medical Institute, Stockholm, Sweden in regard to Atrophic Rhinitis of Pigs are as follows: (1) There is no apparent sex predisposition to the malady; (2) the opinion that atrophic rhinitis is observed only in baby pigs is not well founded—clinical observations in market pigs and older breeding swine indicate that an animal may develop lesions of the disease at any age; (3) first-litter pigs—probably because of unknown genetic factors, and metabolic disturbances —seem to be more susceptible than others; (4) herds that show very rapid growth are more than ordinarily prone to the disease; (5) the changes in the nasal chambers are greatest in their regions anterior to the naso-frontal suture or the regions that have the greatest postnatal growth in length; (6) the writer indicates that pigs may even be attacked during intrauterine life, and such pigs probably die shortly after birth. The author of the article indicates his conviction that bacterial infections frequently play a major role in atrophic rhinitis but that their effect is indirect, by placing a heavy strain on the animal's metabolism. The maladaptation factor appears to be more important than the eliciting pathogen.

*R. R. Dykstra, D.V.M.*

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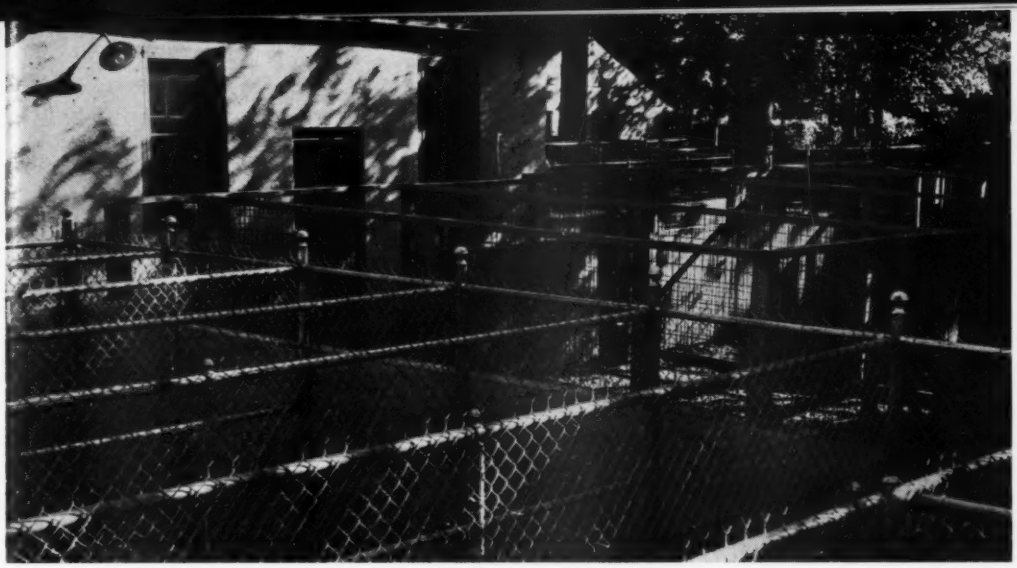

# Early Active Distemper Immunity

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**This clinical trial suggests that active immunization using 2 different types of modified live virus vaccines gave better protection than antiserum alone.**

It is a commonly known fact that humane societies, dog pounds, and, unfortunately, dog hospitals are frequent sources of various contagious diseases, especially canine distemper and infectious canine hepatitis. The problem of early exposure to these diseases is unavoidable in these places. If the young puppies are to be placed in homes as pets, most of them are likely candidates for an early visit to the veterinarian.

The use of antiserum for young puppies in such an environment has been the most common practice in the past. However, there have been repeated failures of this method to protect against the most virulent types of virus. The use of a vaccine to promote early active immunity seemed to be a possible solution to the problem. Such a practice has been reasonably successful in our hospital for some time.

To test this practice more thoroughly, clinical tests were instituted at the local dog shelter where many young dogs are grouped together. It was impossible to have uninoculated controls at the shelter, but for comparison every 3rd puppy was given the usual antiserum while the 1st received egg-adapted chick embryo canine distemper vaccine (Diamond Laboratories) and the 2nd was given ferret attenuated canine distemper vaccine (Fromm Laboratories). A total of 169 puppies were used in this study to calculate the results. However, 54 of these were not seen after they left the shelter and in the results they were assumed to be healthy. Most of the puppies came from rural areas, were approximately 6 to 8 weeks of age and appeared healthy at time of vaccination. They were vaccinated within 24 hours after entering the shelter. Fecal samples and micro-hematocrits were taken on all puppies. This was done to determine their condition at the time of vaccination and to help evaluate the outcome of each puppy. Those with low hematocrits at the time of vaccination were not considered in calculating the results. Each puppy was

tattooed in the ear for identification.

The puppies were classified in 6 groups as to their physical condition in the weeks following vaccination.

I. Those which did not show any symptoms of sickness. These puppies were reported to be clinically healthy at the time of their 1st visit to a veterinarian.

II. Those puppies which were not reported on after vaccination. They were sent out of the shelter apparently healthy, were not taken to any veterinarian nor were they returned to the shelter for any reason.

III. Those that had a fever and a slight diarrhea after vaccination but recovered promptly.

IV. Those that developed a fever which was followed by prolonged respiratory and intestinal infections. Their general condition was unsatisfactory; however, they did recover from their illness without developing neurological symptoms.

V. Those that died from distemper or were euthanized at the request of the owner because of debility and emaciation.

VI. Those whose death was not due to distemper. Two of these puppies died of infectious canine hepatitis, one was injured and the other was euthanized.

## Conclusions

The purpose of this study was to explore the possibility of an improved method of protection for young puppies which are exposed to the street virus of Carre during hospitalization or during their sojourn at a pet shop or animal shelter.

Symptoms of distemper were evident in some of the vaccinated puppies (Group IV). Although these puppies recovered, their sickness was prolonged and the protection could not be considered satisfactory. However, the fact that they recovered indicates that the use of a modified live vaccine is as satisfactory in the over-all protection as the use of serum in such an environment. Note that 10% of the puppies receiving serum developed neurological symptoms, as compared with 2% or 3% of those receiving modified live virus.

More satisfactory immunization was obvious in a greater percent of the puppies which received modified live vaccine prior

**TABLE 1. Comparison of Results Following Vaccination of Young Puppies with Two Types of Modified Live Distemper Virus and Anti-Canine Distemper Serum**

| Group | Total No. | Administered Anti-serum | | Vaccinated with Chick Embryo Origin | | Vaccinated with Ferret Attenuated | |
|---|---|---|---|---|---|---|---|
| | | No. | Percent | No. | Percent | No. | Percent |
| I Healthy | 25 | 5 | 10.8 | 10 | 16.1 | 10 | 16.1 |
| II No report | 54 | 13 | 26 | 18 | 29 | 23 | 37 |
| III Short illness Good recovery | 14 | 1 | 2.2 | 4 | 6.5 | 9 | 14.5 |
| IV Severe and prolonged illness | 56 | 19 | 41.2 | 23 | 45.9 | 14 | 22.5 |
| V Died or euthanized | 16 | 8 | 17.2 | 4 | 6.6 | 4 | 6.5 |
| Neurological symp. | | 5 | 10.8 | 1 | 1.6 | 2 | 3.2 |
| Emaciation and debility | | 3 | 6.5 | 3 | 4.2 | 2 | 3.2 |
| VI Distemper not cause of death | 4 | | | 2 | | 2 | |
| Total | 169 | | | | | | |

to exposure to the natural disease. (Groups I, II and III). The combination of these groups reveal that 40% were protected satisfactorily with antiserum, 51% immunized satisfactorily with chick embryo origin vaccine and 67% satisfactorily with ferret attenuated vaccine.

A considerable number of the puppies which received ferret attenuated vaccine developed symptoms which were diagnosed by veterinarians as those typical of distemper shortly after vaccination, but they responded promptly to treatment (Group III).

Death occurred from infectious canine hepatitis in 2.8% of the puppies that were not protected by antiserum or tissue culture vaccine. The latter vaccine protected all puppies in which it was administered; however, serum also seemed to protect them as well against this disease.

The superior protection with ferret attenuated vaccine over chick embryo vaccine or antiserum while in an environment known to be highly inhabited with the natural disease indicates a sudden response of immunity when a highly potent virus is administered. An early immunity is essential in such an environment.

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## Coccidioidomycosis

A clinicopathologic study of the ocular lesions found in a 7-month-old male Boxer with a concurrent infection of canine distemper and disseminated coccidioidomycosis is described by Robert M. Cello, D.V.M., of Vienna, Austria (formerly of the School of Veterinary Medicine, University of California, Davis).

Simultaneous infection with this organism and the virus of canine distemper has not previously been reported. The study adds the eye to the list of organs which are similarly affected by this disease in the dog and in man.

The existence of a granulomatous uveitis in the case described raises the question of how often such lesions occur in disseminated coccidioidomycosis of dogs. Dr. Cello suggests that the incidence is higher than the literature indicates, since ophthalmoscopic examination of the eye is not done routinely in veterinary practice and lesions which produce no external evidence of their existence usually go unnoticed.

In reviewing the case history it appears likely that the initial clinical signs were manifestations of primary coccidioidomycosis that mistakenly were believed to be those of distemper. This caused the animal to be placed in the infectious ward of a veterinary hospital where it contracted the virus disease. The exact effect which this had on the mycotic infection could not be determined, but it was significant that the rapidly fatal progress of the disease was quite unlike the usual chronic course of disseminated coccidioidomycosis in the dog.

Reed *(Jour. Am. Vet. Med. Assn.,* 128:196, 1958) reported that a 2 to 5 month course was typical of the disseminated infection. The total course in this animal from the first observation of symptoms was 6 weeks. The great number of organisms found in all of the involved organs also was at variance with the findings in previous cases necropsied at Davis. Dr. Cello concluded, therefore, that the resistance of the animal to *C. immitis* was decreased by the concomitant viral infection, resulting in a more extensive and rapid invasion than usual.

There appeared to be no direct relationship between the 2 agents in the eye. The coccidioidal infection was localized in the uveal tract and involved neither the conjunctiva, which contained numerous distemper inclusions, nor the areas of the retina in which this virus usually is found. This led the investigator to the conclusion that the extensive ocular lesions were the result of the enhanced dissemination of *C. immitis* rather than any specific action of the distemper virus.

*Arch. Ophth., 64:897-903 (December) 1960*

# Early Distemper Protection for Exposed Dogs

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Early protection of exposed pups has been achieved with a tissue culture vaccine which tends to confirm the observations of Johnson and Bredahl that active immunity is superior to antiserum protection.

The practitioner of small animal medicine is constantly confronted by problems concerning immunization programs for dogs. Among the most familiar of these is the proper procedure to follow in the case of puppies which may be exposed to distemper. Puppies originating in breeding kennels which have histories of enzootic distemper infection, or those sold through pet shops and animal shelters are of this category. As a class, these puppies are notorious for causing grief and needless expense to their new owners and misunderstanding or ill-will between veterinarians and the operators of these establishments. The triggering mechanism is usually a break in the distemper immunizing procedure used, with unpleasant consequences for all concerned.

Similarly, the veterinarian must satisfy himself that he is providing adequate protection to dogs prior to their known or presumed exposure to distemper. Problem situations include: field trials and dog shows, hospitalization and boarding of dogs in veterinary hospitals, introduction of new stock into breeding kennels and research animal colonies, and incarceration in dog pounds. If left unprotected, susceptible puppies can almost invariably be counted upon to become infected with the canine distemper virus.

The methods which are adopted to meet these varying conditions must differ with the situation and with the judgment of the veterinarian. Canine distemper antiserum and antibody concentrate are commonly employed to cope with such exposures, and these products have a very definite place in the veterinarian's armamentarium.

Used alone, these passive agents may not be sufficient to provide protection. In a recent report on various immunization procedures used on dogs originating from animal shelters, Johnson[3] contends that he was able to prevent losses from canine distemper in only about 40% of such puppies by the use of canine distemper antiserum. Using various modified live virus vaccines of chick embryo and ferret tissue origin, he was able to prevent such losses in up to 67% of puppies in a parallel study.

Recently, considerable animal experimentation was performed during the clinical evaluation of a new modified live virus canine distemper vaccine of tissue culture origin (Tempacine, Corn States Laboratories). This work has disclosed valuable information on the rapidity with which protection against a severe distemper virus challenge can be established. Another series of studies indicated that a vaccine of high potency can successfully immunize susceptible dogs if administered simultaneously with antiserum or antibody concentrate. These findings offer the promise of providing rapid and complete protection against canine distemper infection.

The intracerebral challenge technique was adopted for use in routine potency tests. This is the most precise and rugged method known of evaluating protection afforded by distemper products.[4] Continuing experience in the testing of production lots of this vaccine has not only established confidence in the reliability of the challenge and the efficacy of the product, but has also developed information of interest relative to the early protection afforded by the vaccine. Routine potency tests of the vaccine are performed by challenging vaccinates 7 days following vaccination with fractional doses. In tests to date, 41 out of 42 vaccinates have survived the intracerebral inoculation of the virulent Snyder-Hill strain of canine distemper virus, while 31 out of 34 unvaccinated controls have died. In almost all cases, protection occurred in animals before measurable amounts of canine distemper virus neutralizing antibody could be detected.

These results led naturally to speculation as to the earliest point in time when protection would appear. Accordingly, series of experiments were performed with results as summarized in Table 1.

All dogs participating in the experiments summarized in the tables and in the potency tests previously mentioned had titers of canine distemper virus neutralization of less than 1:40, as determined by the method of Burgher, Baker, *et al.*[4] When possible, littermate puppies were used. The challenges used were: 0.25 cc. of a 1:100 dilution inoculated intracerebrally or 1.0 cc. of a 1:10 dilution inoculated intramuscularly of Snyder-Hill strain canine distemper virus.

**TABLE 1**

**Summary of Early Protection Studies Using Modified Live Virus, Tissue Culture Origin Canine Distemper Vaccine**

| Day of Challenge | Vaccination Route | Challenge Route | No. Survived/No. Challenged |
|---|---|---|---|
| 0 (Simultaneous) | Intramuscular | Intracerebral | 0/2 |
| 0 | Intravenous | Intramuscular | 2/3 |
| 1 | Intramuscular | Intracerebral | 1/6 |
| 2 | Intramuscular | Intracerebral | 11/13 |
| 3 | Intramuscular | Intracerebral | 3/3 |
| 3 | Intravenous | Intracerebral | 2/2 |
| 4 | Intravenous | Intracerebral | 8/8 |
| 5 | Intravenous | Intracerebral | 2/2 |
| 7 | Intravenous | Intracerebral | 2/2 |
| 7 | Intramuscular | Intracerebral | 9/9 |

36 uninoculated controls were used, of which only 2 survived.

**TABLE 2**

**Summary of Protection Studies Using Modified Live Virus, Tissue Culture Origin Canine Distemper Vaccine Administered Simultaneously with Passive Antibody**

| Vaccine Route | Amount Trivalent Antiserum Per Pound Body Weight | Amount Antibody Concentrate Per Pound Body Weight | Challenge Results (14 Days Post-Vaccination) No. Survivors/ No. Challenged |
|---|---|---|---|
| Intravenous | 1.0 cc. | | 2/2 |
| Intramuscular | 1.0 cc. | | 2/3 |
| Intravenous | 2.0 cc. | | 1/1 |
| Intramuscular | 2.0 cc. | | 2/2 |
| Intravenous | | 0.1 cc. | 2/2 |
| Intramuscular | | 0.1 cc. | 2/2 |
| Intramuscular | | 0.2 cc. | 2/3 1 Trauma Death |
| Intramuscular | 2.0 cc. | 0.1 cc. | 0/1 |
| Unvaccinated contact controls | 2.0 cc. | | 0/3 |
| Unvaccinated contact controls | | 0.1 cc. | 0/3 |

The severity of the challenge used in these early protection experiments may have masked the possibility of even earlier protection if a less severe challenge, mimicking natural exposure, had been used. When the route of vaccination and challenge was varied, definite indications of protection were demonstrated in the simultaneous challenge experiments. Because natural infection usually results from the naso-pharyngeo-tracheal inoculation of a minimal dose of virulent virus, it is reasonable to speculate that protection against this presumably less severe exposure may appear very early. Further investigations in this area are presently in progress at Corn States Laboratories.

Another fruitful line of investigation were the studies which determined that protection and serological evidence of immunity could be established in dogs when this highly antigenic vaccine under study and potent passive protective agents were administered simultaneously. A similar experiment has been previously reported.[1]

Serum or antibody concentrate was administered to the unvaccinated contact controls in the experiment. Examination of post-vaccinal serum samples taken at the time of challenge indicated that the dogs which had received both vaccine and passive protective agents had acquired significant antibody titers as well as protection against a severe challenge. Unvaccinated controls which had received the passive agents without vaccine had no detectable titers in 14 days, and were entirely susceptible to challenge at this time.

## Summary

In a series of experiments in susceptible dogs vaccinated with a tissue culture origin modified live virus canine distemper vaccine, unexpected early protection against a severe challenge was noted. Simultaneous use of passive immunizing agents did not affect the antigenic capacity of this vaccine. These findings point the way toward a more effective means of protecting susceptible puppies both before and after presumed exposure to natural distemper infection.

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# Case Report: Piperazine Citrate Tests

It has long been the hope that some drug could be found which, when mixed with food or dissolved in drinking water, would eliminate all of the intestinal parasites of dogs. Some extravagant claims have been made for piperazine in several forms, especially that it is more efficient in removing hookworms than early work indicated.

Fecal examinations had indicated it was time to deworm the dogs in our kennel. It was decided to investigate the possible effect of long-term use of piperazine citrate to determine if it was more efficient than the 1 or 3 day dosing currently being recommended. There was no opportunity to study its effect against tapeworms; the kennel was free of them.

A Beagle hound had been dosed for 11 days at the rate of 500 mg. of piperazine citrate per 5 lb. of body weight, double the recommended dose. After a week's rest, the dog received 11 more days of treatment before it was sacrificed and the liver studied for damage by a competent pathologist. There was none.

The infected runs in the kennels held 86 mature dogs and 136 puppies. Fecal examinations of 50 puppies showed no parasite ova. The piperazine citrate was administered by dissolving it in the drinking water in which the food was mixed, thus giving an even distribution. Dosage was calculated to supply each dog with 250 mg. per 5 lb. of body weight. The treatment was continued for 10 days.

The presence of the drug in the food did not make it unpalatable and food consumption did not decrease. Slightly more food was consumed at the end of the 3 days than had been eaten at the start, probably explained by the fact that many of the animals were growing puppies.

Fecal examinations were conducted by pens. At the conclusion of the treatment period, another set of fecal examinations was made. All fecal examinations were centrifuged, sodium nitrate flotations using a 1.4 sp. gr. solution.

Six pens were studied by daily fecal examinations. Composite samples were collected about ½ hour after the morning meal. A small sample was taken from each of the stools found and mixed together. Each run contained litters of 3 to 6 puppies or 2 grown dogs. The dogs in each run had been raised together, dewormed at the same time and fed the same food. Parasite infection is usually fairly uniform in such dogs, but occasionally one finds variations. These differences must be allowed for. More of the feces of 1 dog than of another may have been gathered or possibly 2 stools from the same dog sampled.

Ascarids were found in the feces 24 hours following treatment. Some dogs continued to void them for 3 days. No hookworms were found. Fecal examinations at the conclusion of the study revealed a complete elimination of ascarids. As many hook-

worm eggs were found in some pens at the end of treatment as there were at the start, in some cases a reduction and in some an increase, which could be explained on the basis of composite samples. There had been no effect on whipworms, some runs showing an increase in numbers of eggs.

The daily fecal examinations on the 6 pens, each containing 2 grown or nearly grown dogs, indicated the progress of the study and showed that 3 days of dosing at this level of the drug were necessary to eliminate ascarids.

### Conclusion

Piperazine citrate, administered in dogs' food at the rate of 250 mg. per lb. body weight and given for 10 days, was effective in ridding dogs of ascarids but not the southern hookworms or whipworms. At least 3 days' dosing is necessary to eliminate the ascarids at this level of administration.

*Leon F. Whitney, D.V.M.*
*Orange, Connecticut*

*Branchial Cyst. In contrast to salivary cyst which appears suddenly and fills rapidly, this often does not show much distention until the 2nd or 3rd year. (Photo by J. Mosier, D.V.M., Kansas State University.)*

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# Management of Cardiac Arrest

The primary consideration in the treatment of cardiac arrest is the establishment of adequate oxygenation of cerebral tissues, myocardial tissues and the body tissues in general. Oxygen tension in the capillaries must be increased in order to return to the heart and brain tissues their normal excitability. There are 2 ways in which to accomplish this. One is by cardiac massage and the other by intra-arterial transfusion under pressure.

After the diagnosis of cardiac arrest has been made, oxygen must be supplied to the blood. The most effective means of supplying this oxygen is by positive pressure through an endotracheal catheter.

While a patent airway is being established, the chest should be opened and the heart exposed. There is no time to prepare the thorax for aseptic surgery. A quick, bold incision must be made. Care should be taken not to cut or tear the lungs during the thoracotomy.

The heart may or may not be removed from the pericardium, but massage will be more effective when the heart is removed from the pericardium and, therefore, has a chance to fill to its fullest during diastole.

Cardiac massage should begin immediately, and the heart should be rhythmically compressed and decompressed by the hand

of the operator. The ideal rate for massage is in the range of 60 beats per minute. This soon becomes tiring to the operator, and a more practical rate of 30 to 40 beats per minute can be adopted.

If available, intra-arterial transfusions under pressure may be given. This will tend to circulate blood into the myocardium through the coronary system and likewise will furnish oxygenated blood to the ischemic cerebral tissues.

In considering various drugs to be used, one prime thought must be kept in mind: the drug must be used in proper dosages and must be used specifically. Drugs should not be given immediately, but the heart should be massaged for at least 3 minutes prior to the administration of any therapeutic agents.

Epinephrine is a drug frequently thought of as specific for cardiac stimulation; however, it should be kept in mind that epinephrine will shorten the refractory period of the heart making it very susceptible to fibrillation. When used, it should be diluted 1:10,000 rather than the commonly available 1:1,000 dilution. Three to 5 cc. of the 1:10,000 dilution should be injected into the left ventricle. This injection will be more effective in the left ventricle as there is immediate perfusion through the coronary system.

The effect of the calcium ion upon the arrested heart has been shown to be of great significance. The tissues damaged by hypoxia release considerable potassium. The elevated potassium level in the blood stops the heart in diastole. Calcium ions can be administered to neutralize this effect. Calcium gluconate, 3 cc. to 5 cc. of a 10% solution, can be given, if deemed necessary. Care must be taken not to give an excess of calcium ions as this will also stop the heart. Other drugs that may be effective include the various vasopressive agents, such as mephenteramine sulfate and nor-epinephrine.

The most accepted treatment for fibrillation is the use of electrical shock in conjunction with cardiac massage. Several defibrillating apparatuses have been described. The electrodes should be applied to the surface of the heart over saline-soaked gauze sponges to prevent excessive burning of the myocardium in the area of contact.

Rubber gloves must be used to prevent electrical shock to any personnel in contact with the animal. A rapid make-and-break circuit is used to furnish 0.5-1.5 amperes of current. Each individual shock should be no longer than 0.25 seconds and should not be more frequent than 3 or 4 at a time. Although electrical shock will usually stop fibrillation, it must be borne in mind that cardiac massage should always be used in conjunction with the shocking. Should fibrillation return, the heart may again be electrically defibrillated.

After establishment of an effectual heart beat, the aorta may be compressed distally to the origin of the brachiocephalic artery to increase the blood flow to the vital nervous centers. Anoxia may continue in other tissues of the body for a longer time than in these vital centers.

The beating heart should be closely observed for 10 to 15 minutes to be sure it does not revert to its previous state. During this observation period, the thoracotomy incision can be cleaned in preparation for closure. A saline lavage containing an antibiotic should be used in the thoracic cavity. Systemic antibiotics should also be given. If it is necessary to give parenteral fluids, it must be remembered that the heart is still in a weakened state and that the circulatory system should not be overloaded.

When the operator feels that an effectual heart beat has been established for an adequate length of time, the thoracotomy may then be closed. The animal should be observed for the remainder of the critical post-operative period, epsecially the first 8 hours.

*J. Kling, A. Hahn, R. Horne, The Auburn Veterinarian, Vol. XVII, No. 1, pp. 17-22.*

## *Medical Interchange*

*Notes and abstracts screened from the medical literature for their special interest to veterinarians by Lynn Stratton Morris.*

# Parenteral Burn Therapy

A single intravenous solution containing 0.66% sodium chloride, 2% protein and 5% glucose offers certain advantages for the parenteral fluid therapy of patients with 2nd and 3rd degree burns involving 15% or more of the body surface.

This solution replaces water, salt and protein continuously at the same rate at which they are being lost from the circulation. Previous regimens replaced these factors discontinuously by alternate infusions of plasma, saline, or dextrose and water. The greatest advantage of this simplified regimen lies in ease of management and the virtual absence of fluid and electrolyte problems.

Drs. J. Frederick Eagle, Jr., of St. Luke's Hospital, New York, and Worthington G. Schenck, Jr., and Walton Shim, of the Edward J. Meyer Memorial Hospital, Buffalo, reported that this treatment has been used for all burn patients admitted to their respective hospitals during the past 4 to 6 years.

The method is recommended for the first 48 hours following the burn. Replacement of red blood cells also is necessary, but seldom during this early period.

The initial rate of infusion for adults is 20 ml. per hour for each percentage point of body surface burned. If the onset of treatment must be delayed, the starting rate should be increased so that the patient will have received 160 ml. per percentage point of body burn 8 hours after the burn. After adequate urine flow is established, the infusion rate is gradually reduced according to the rate of urine production.

Starting rates for children must be calculated according to weight and surface area. The following formula is suggested for calculating the surface area of the infant:

$$\text{Surface area} = \sqrt[3]{(\text{weight, kg.})^2} \times 0.1$$

The calculations for fluid intake during the first 48 hours are: (1) 30 ml. per square meter for each percentage point of body surface burned (up to 50%) to replace fluid lost through the wound, plus (2) an amount of fluid equal to 10% of the body weight in kilograms to account for edema accumulation, plus (3) 2,000 ml. per square meter for insensible water loss, plus (4) 2,000 ml. per square meter for adequate urinary output.

One-third of this fluid usually will be required in the first 8 hours after the burn, and the initial rate of infusion should be adjusted to achieve this aim. As soon as

urinary output exceeds 40 ml. per square meter per hour, the rate of infusion should be decreased in the same way it is for adults. For patients with burns of less than 15% to 20% of the body surface area, in whom edema is not a major factor, the fluid equal to 10% of the body weight in kilograms, (2) above, need not be given.

Results suggest that this regimen will lower mortality during the first 48 hours.

*Jour. Am. Med. Assn. 174:1589, (Nov. 19), 1960*

## Topical Wound Therapy

Topical application of penicillin spray and of bacitracin-neomycin spray as the sole treatment of a lethal, massive open wound of the goat thigh were of significant benefit in prolonging survival time.

Major Janice A. Mendelson, MC, of the Trauma Investigation Branch, Biophysics Division, Directorate of Medical Research, United States Army Chemical Research and Development Laboratories, Md., reported on an evaluation of various types of topical therapy for massive wounds. Development of simple expedient therapy to allow prolongation of the interval between wounding and definitive surgical care was the purpose of the study.

Penicillin spray was the only substance tested which produced significant benefit when applied as a single treatment 5 hours after injury. Four out of 5 animals in this group lived over 40 hours; none of the control animals lived as long as 36 hours. Mean survival time thus was approximately doubled when a single application of penicillin spray was given either immediately after injury or 5 hours later. Prolongation of survival time was even greater with repeated penicillin treatment.

Observations were made on a total of 115 goats. The substances tested were applied as a single instillation or spray immediately or 5 hours after injury, and as repeated instillations or spray, starting either immediately or 5 hours after injury and continued twice daily as long as the animal survived. Only the bacitracin neomycin spray (25,000 units of bacitracin and 250 mg. of neomycin) and penicillin spray (2 million units of crystalline penicillin) in 25 ml. volumes showed significant prolongation of mean and median survival times. Both solutions were applied as a spray by means of the DeVilbiss atomizer with 10 pounds of pressure.

Prolongation of survival time showed a significant correlation with retardation of growth of *Cl. welchii*. In a significant number of instances it was possible to predict survival time by means of a formula based on clostridia count alone. Addition of the *Staphylococcus aureus* and *Staphylococcus albus* factors added essentially nothing to the predictability.

Dr. Mendelson pointed out that there is no evidence as yet that any topical application by itself will produce as good results as adequate removal of debris and devitalized tissue. The only implication of the current studies is that topical therapy may prolong the period during which debridement may be effective. Secondarily, it may reduce morbidity from wounds as well as the number of deaths.

Because longest survivals were obtained in those few animals which accidentally acquired infestation with maggots, the research laboratories are studying the topical use of enzymes and other substances, hoping to improve on the "maggot therapy."

*Surgery, 48:1035-1047 (December) 1960*

# The Fluorescent Rabies Antibody Test

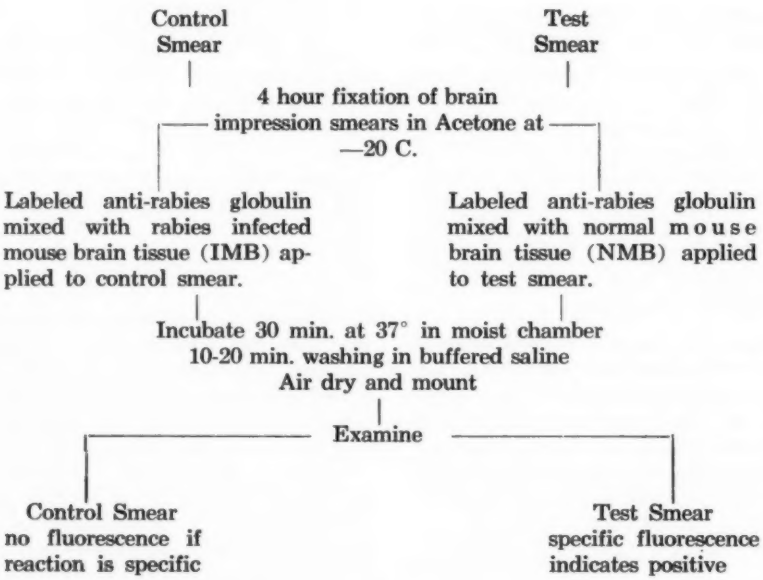
The fluorescent rabies antibody test may be used to advantage to help define the antigenic nature of atypical or otherwise questionable inclusion bodies observed during direct microscopic examination, report James L. McQueen, D.V.M., Arthur L. Lewis, D.V.M., and Nathan J. Schneider, Ph.D., of the Florida State Board of Health.

Based on the results of routine examination of specimens received at the central laboratory during a 13 month period, the investigators believe the FRA test to be a valuable addition for the rabies diagnostician. Some limitations also are discussed.

The FRA test was in complete agreement with the mouse inoculation results on a total of 825 fresh or frozen specimens, whereas the results of the Negri examinations gave only a 98.3% correlation. The FRA test detected all of the 70 positives included in the group. In contrast, in only 66 of 70 positives, or 94.3%, were Negri bodies demonstrable.

Preliminary studies indicate the FRA test to be highly specific. It appears to be more sensitive than the direct microscopic examination of Sellers' stained slides; however, the authors explain, additional studies are needed before any firm conclusions can be drawn on the sensitivity of the FRA test as compared to standard mouse-inoculation technics.

The procedure for staining rabies virus antigens with fluorescent tagged antibody in the brains of naturally infected animals is represented schematically.


Control Smear
Test Smear
4 hour fixation of brain impression smears in Acetone at —20 C.
Labeled anti-rabies globulin mixed with rabies infected mouse brain tissue (IMB) applied to control smear.
Labeled anti-rabies globulin mixed with normal mouse brain tissue (NMB) applied to test smear.
Incubate 30 min. at 37° in moist chamber
10-20 min. washing in buffered saline
Air dry and mount
Examine
Control Smear no fluorescence if reaction is specific
Test Smear specific fluorescence indicates positive

Fluorescent Rabies Antibody Test (Direct Method)

Results would indicate that fragments of brain tissue preserved in glycerol-saline were not satisfactory for FRA examination. Glycerination even for short periods in almost every case resulted in a material and often a diagnostically critical quantitative and qualitative decrease in visible fluorescence.

One of the most distressing limitations to routine use of the procedure at this time is the unavailability of commercially produced reagents. At present there is no dependable commercial source of labeled antiserum. Moreover, it is in the production of a satisfactory conjugate that the major obstacle to successful performance of the test is encountered. The authors have succeeded in producing satisfactory conjugates 5 out of 7 times, using 6 different lots of antisera and 4 different lots of fluorescein isothiocyanate obtained from 2 sources. It was apparent that some lots of dye gave better conjugation of the protein than did others.

In addition the authors stressed that the success obtained with the technic may depend largely on the conscientious application of certain prescribed procedures in the preparation of fluorescent conjugates and in the performance of the test.

*Am. Jour. Pub. Hlth., 50:1743-1752 (November) 1960*

## Wild Life Rabies Cases Decrease

As the incidence of rabies in domestic animals has decreased, reported cases of rabies in sylvatic animals have sharply increased, report Drs. John R. Kent and Sydney M. Finegold, of Los Angeles.

There has been a definite and striking increase in recent years in the number of human cases of rabies caused by the bites of rabid wild animals, chiefly foxes and skunks. Six cases transmitted by the bites of brown bats also have been reported in the medical literature. During the past several years, wildlife sources have accounted for 20% of human deaths from rabies in the United States. Wild-animal rabies also means huge livestock losses and constitutes a serious reservoir of infection for dogs.

Anyone bitten by a bat should be treated promptly. The apparent health of a bat-bite victim is no assurance of freedom from rabies, since the survival period for insectivorous bats with the natural infection is not definitely known. Every effort should be made to submit the bat for studies to detect rabies. Only 50% of rabid bats show Negri bodies and laboratory diagnosis may take longer than the incubation period of rabies in man.

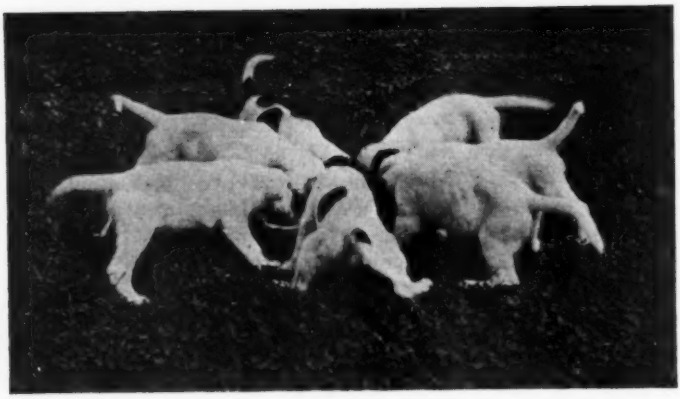
There is no treatment available for rabies; since the disease is 100% fatal in human beings, prophylaxis, or postexposure treatment, is extremely important.

Unpublished data indicate that duck-embryo vaccine behaves as well as the Flury vaccine in protecting dogs from challenge with virulent street-rabies virus. In 1 study, 850 patients with rabies exposure were treated with the duck-embryo vaccine (no antiserum) without ensuing rabies. Greenberg and Childress report on reactions and antibody titers in 123 patients given duck-embryo rabies vaccine and 127 given Semple vaccine. No general reactions occurred with the former, but there were 2 cases of encephalomyelitis in the latter group.

The duck-embryo vaccine has been found to give excellent recall in persons previously immunized with Semple vaccine. This may be very important for persons treated some time ago and then re-exposed to rabies, since such persons are particularly susceptible to encephalomyelitis if retreated with Semple vaccine.

[Requests for reprints should be addressed to Dr. Finegold, Wadsworth Veterans Administration Hospital, Wilshire and Sawtelle Boulevards, Los Angeles 25, Calif.]

*New Eng. Jour. Med. 263:1058, (Nov. 24), 1960*



# Canine Pediatrics

**Part 2**

**The first installment was published in the February 1961 issue.**

The young puppies need between 60 and 70 calories per lb. per day during the initial feeding period. Although bitch's milk provides from 90 to 130 calories per lb. per day, the 60 to 70 calorie level was found to represent the optimum mean for adequate nutrition and compatibility during this feeding period. The puppies made adequate growth and development at this level with total absence of any pathological conditions.

The normal fluid requirement of puppies ranges from 2½ to 3 ounces per lb. per day. The percentage concentration of the formula is lowered during this initial period in order to maintain proper fluid intake at this low caloric level. Since the caloric level increases more rapidly than the fluid requirement, the concentration of the formula is increased as the feeding progresses.

*Feeding Regimen.* Three feedings per day at 8 hour intervals have proved to be optimum.[3] Most recommendations for feeding newborn puppies specify every 4 hours, missing 1 feeding during the night. The 8 hour interval is believed to allow adequate time for complete digestion and assimilation of feedings. Also, this interval allows the puppies more time for rest and rehabilitation of tissues. Such a schedule requires much less time and expense.

The puppies should be awakened at exactly 8 hour intervals, their backs and sides massaged to stimulate the muscles and skin. This improves their tone and development. It also helps to satisfy the puppy's desire for companionship.

The formula should provide 60 to 70 calories per lb. per day during the 1st and 2nd weeks, 70 to 80 the 3rd week, 80 to 90 the 4th week and 90 to 100 the 5th week.

During the 5th week, 1½ teaspoons of dry dog meal should be added to each 2½ ounces of milk formula, or about ½ teaspoon per feeding. This should be increased at the rate of about ½ teaspoon every 4 to 5 days. The formula used may be any of the simulated bitch's milk products that are on the market today or a home formula as suggested in *Canine Medicine,* 2nd Edition, will be very satisfactory.

The following feeding directions are recommended:

1. Only the quantity of formula required for a 24 hour period should be prepared at one time. Refrigerate the portions that are not used until the next feedings.

2. Warm the formula mixture to approximately body temperature (100 F.) before each feeding.

3. Very young puppies will have to be fed from some type of bottle which will satisfactorily take a nipple. A standard infant-feeding nipple is suitable for larger breeds of puppies but a regular puppy nipple will be better for the smaller breeds.

4. The hole in the nipple must not be too large. It should be sufficiently small so that the puppy has to suck with some vigor. This not only prevents choking but also prolongs the feeding period.

5. The bottle should be held so that the puppy does not ingest any air with the formula. If this occurs, the puppy may regurgitate in an effort to expel the air or if it is retained, it may cause the puppy pain.

6. The puppy should be taught to lap out of a dish and the bottle discarded after a period of 4 to 5 weeks before any solid food is added to the ration. They can best be taught to lap by wetting the little finger with some formula and while the puppy is sucking, gently drawing its mouth into the dish. Its head should be held with the other fingers of the hand and its paws with the opposite hand. The puppy should not be excited or forced during this procedure. In this way the puppy will learn very rapidly to lap out of a dish in a satisfactory manner.

7. The equipment and utensils must be kept clean and sanitary at all times. After each feeding the bottles, nipples and other utensils must be thoroughly washed in hot, soapy water and rinsed in hot water.

8. After the puppies have learned to lap and to eat semi-solid foods, the amount of dry meal added to the feedings should be increased. By the time the puppies are about 8 weeks old, the amount of dry food added should be such that the feeding mixture is of a mushy consistency. There should be adequate bulk in the diet at this time for proper development of the digestive system. After the puppy has taken the mushy mixture for several days, a small quantity of finely ground, lean beef should be mixed with each feeding.

9. At no time during the feeding period should any attempt be made to accelerate an already satisfactory rate of growth and development by feeding excessive amounts or concentrations of formula. Any rapid change in the amount of concentration of the formula will cause digestive upsets that are difficult to arrest.

## Clinical Observations

Important data regarding the progress and physiological condition of artificially-fed puppies can be obtained from a number of standard clinical observations. These observations should be a routine practice in canine pediatrics.

The simplest observation to be made and recorded is the daily weighing of individual puppies. This is also one of the most valuable observations. Any change or lack of change in weight is an index to the progress and development of the puppy. A steady gain in weight with a normal fecal condition means a healthy puppy. Stationary weight without any abnormal fecal condition indicates subnormal metabolism and inadequate nutrition. Stationary weight or declining weight with abnormal fecal condition indicates an acute dietary disturbance or a possible gastrointestinal infection.

Some puppies of the litter may double

their weight in 10 days, but in a large number of cases this rate of gain will not be realized. The ideal rate of development of an artificially-fed puppy should be constant and uniform without putting on an excessive amount of weight.

Young puppies should be weighed at the same time each day. They tend to lose a slight amount during the night. If they are weighed immediately after eating at one time and not at the next, this will not be an accurate index of progress. A beam type scale is recommended because it is more accurate. The scale should read in gm. and be sensitive to 1/10 of a gm.

Daily observations of the condition of the feces are also of great importance. Normally, the puppy's feces are brown in color and reasonably well formed. They are easily evacuated and the puppy is clean around the rear quarters. Normal feces indicate a proper functioning intestinal tract and proper colonic reflexes.

A disturbance in the diet is followed by a sequence of changes in the color and physical characteristics of the feces. The first change is to a light brown color and semi-formed condition of the feces. Next, it progresses to greater fluidity and has a greenish cast. The color then diminishes to a light green, to a dirty grey and finally to a dirty white. The latter indicates the evacuation of undigested food.

Abnormal conditions of the feces parallel a breakdown in the muscular tone of the colon and the entire intestinal tract. In extreme cases the voluntary process ceases entirely.

During this time the feces should be checked for intestinal parasites. Since puppies are infected *in utero* by ascarids and hookworms if the mother is infected, the puppy's feces should be checked at 2 week intervals.

Milk of magnesia is especially beneficial in correcting abnormal fecal conditions caused by dietary disturbances. A dose of ½ a teaspoon for a very young puppy, followed by a small amount of sterile water, administered at one of the regular feeding periods in place of the formula, aids in correcting the condition. It is advisable to reduce the quantity of formula for the next 1 to 2 succeeding feedings. For larger puppies it will be necessary to increase the quantity of milk of magnesia.

The skin of the puppy is an index of its health. It can be tested for turgidity and elasticity simply by pulling it out with the thumb and forefinger. If the puppy is in good health, it will snap back quickly. If it remains pinched up or goes back slowly, it is a sign of dehydration and subnormal health.

Glossy, silky hair of fine texture is characteristic of a well-nourished, thriving puppy. The hair of an unhealthy puppy is coarse, matted and straggly.

During these examinations of the skin and hair, the attendant should watch for external parasites. Lice or fleas may have been gotten from the mother and very rapidly become a serious problem on young puppies. If any are found, only rotenone preparations should be used on the puppies.

The baby puppy's biological and environmental needs are similar to those of the human infant. It responds in terms of growth and health to care and devotion. It suffers parallel physiological setbacks as a result of neglect.

The successful raising of artifically-fed puppies is not an easy task. It requires much patience and constant attention to details. However, it is not impossible and the reward of helping an owner successfully raise orphan puppies is the satisfaction of having served your client in a way you were trained to do.

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# Hemolytic Diseases of Newborn Pigs

A. H. QUIN, D.V.M.

Sudden deaths of entire litters of baby pigs occur each year in every hog raising area. Some of these may be cases of hemolytic disease. In this paper, Dr. Quin gives a concise review of the disease and a postulation of the mechanism of action of this disease, supported by Hungarian authorities.

This condition, which may result in a 100% death loss of individual litters, is somewhat comparable to the Rh incompatibility blood type factors of human erythroblastosis fetalis or to the ictero-anemic syndrome in newborn foals.

It has been established that there are 4 classic blood types in swine, types A, B, C and D. In the living animal the red blood cells contain a specific type antigen and the blood plasma contains the specific antibody against it.

The etiological concept of hemolytic disease of newborn pigs is that if the sow whose red blood cells do not contain the given antigen is fertilized by a boar whose red blood cells do contain the given antigen, the pigs *in utero* may sensitize the sow to

the red cell antigen inherited from the boar. The sow responds by developing a high antibody titer against the specific sensitizing antigen.

There is one striking difference in comparing the human Rh blood type syndrome with hemolytic disease of newborn pigs or postnatal icteroanemia of baby pigs or foals. The human mother has a 3 layer placenta, the hemochorionic placenta. Both swine and mares have a 7 layer placenta or epithelio-chorionic placenta. In the 3 layer placenta of the human mother the sensitizing antigen is absorbed into the maternal circulation, and mass iso-agglutinating antibodies are passed back through the placental barriers to the fetus. As a result, there is a gross linkage or binding of the maternal antibodies with the red cell antigen of the fetus and frequently fetal death.

In sows and mares with 7 layer placentas the sensitizing antigen, being of small molecular size, passes through the placental layers into the circulation of the sow or mare. It stimulates production of antibodies against the boar-type red blood cells inherited by the young *in utero*.

But here is the important difference. The antibodies formed by the sow or mare are too large a molecular size to pass back through the 7 layer placenta to the fetuses *in utero*, and no fetal death results. This explanation is a postulation of the Hungarian authorities.[1]

## Clinical Symptoms

As a consequence, the pigs are born at full term strong and healthy. They take the first milk with colostrum of the sow, which is rich in the antibodies or iso-agglutinins. During the first 24 to 36 hours these antibodies are absorbed by the digestive tract. The colostral antibodies bind with the paternal antigen in the red blood cells of the pigs, the erythrocytes are destroyed and death by hemolysis and icterus occurs within 24 to 48 hours.

Hungarian authorities cite occurrence of naturally occurring hemolytic disease on 2 hog farms involving 60 litters of both pure and crossbred stock. At farm A the litters of 13 out of 52 sows were affected, with a death loss of 46 pigs or 67.5% of the affected litters. At farm B the litters of 19 out of 70 sows were affected, with a death loss of 82 pigs or 73.5% of ill pigs. Of the remaining 28 litters, 209 of the total 324 pigs, fell victims to hemolytic disease.

The clinical symptoms cited by the Hungarian authorities are of most diagnostic value. The piglets are well developed, healthy at birth and suckle normally. Yet later, and invariably within the first 48 hours of life, most of them stop sucking, become disinterested, hide in the straw, tremble, stagger, bristle their coats and squeal. On closer inspection jaundice of the skin and membranes becomes evident even in the non-white breeds. The yellowish discoloration is most conspicuous in the axillary and inguinal regions, while that of the membranes is best seen by opening the mouth or by examining the conjunctiva.

The urine is red or coffee-colored, as it contains much hemoglobin and protein. By mildly pressing the bladder, it is not infrequently possible to obtain urine from the live piglet sufficient to establish the hemoglobin content, thus confirming the diagnosis.

The blood drawn from diseased piglets does not coagulate; it is lacquer-like in its entirety, scarlet in hue and with a yellowish iridescence at the margins. The amount of sediment obtainable from centrifugation is moderate. In bloodsmears hardly any normal erythrocytes are encountered; most of them are in a state of dissolution.

The course of the disease is rapid. Most of the affected piglets die. Those showing symptoms in 12 to 36 hours after farrowing die in another 6 to 36 hours. Death usually occurs on the 2nd day, less frequently on the 3rd, and very seldom on the 4th or the end of the 1st day.

The entire or almost the entire litter

is affected. The explanation of why only 67.5% and 73.5% of the piglets on the farms had died seems to be that on these farms it was the custom to have each sow mated to 2 different boars. Apparently, only one boar had an antigen different from the sows.

### Lesions

On post-mortem examination the most prominent lesion is jaundice of the skin and all internal surfaces but particularly marked in the subcutaneous connective tissue. The blood is scarlet, thin and does not coagulate. In the body cavities and pericardial sac an excess of reddish colored transudate may be noted. Port wine to rust colored urine is present in the bladder. The kidneys are russet or chocolate brown. The liver is slightly enlarged, brittle and a yellowish to yellowish brown color.

### Discussion

It has been stated by several research authorities[2] that isoimmunization or hemolytic disease occurs only in sows previously vaccinated with blood-tissue origin, crystal violet hog cholera vaccine or after parenteral injection of incompatible boar red blood cells.

The author believes that the disease can appear without history of previous injection of sows with vaccines of blood origin. Some years ago a herd of several big-type Poland China sows lost the entire litters of 2 successive farrowings when crossed with a Tamworth boar. On the same farm other sows bred to a Poland China boar all had normal litters.

Cases of hemolytic disease have been reported in Hungary from litters of sows which were known to have never been injected with swine serum or vaccines of blood origin.[4] One of the first cases described by these workers in 1952 occurred in a sow reared at the experimental institute. It was known that the only injection this sow had received was a vitamin A preparation. More recently they have observed cases of hemolytic diseases of piglets in crosses of Mangalica sows (Hungarian breed) and Berkshire boars. Some cases have occurred at an experimental farm where no serum or vaccines of blood origin were used.[3]

Hemolytic disease is not an economically important disease to the swine industry. The number of young pigs lost each year from it is quite low, yet probably some cases occur that are not reported by the swine raisers because of the sudden loss but small number of litters involved.

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## Detection of Heat, a Problem in Swine Artificial Insemination

A difficulty encountered by research workers in the field of artificial insemination of swine is that in gilts the heat periods are frequently not clearly demonstrated. It has been suggested that vasectomized boars be kept with the females to detect estrus. Because of the difficulty in detecting estrus based on the females' visible reactions, the settling by artificial insemination of gilts is about 50%, and in sows 57%. Approximately normal conception rate following natural service is close to 70%. One collection of boar semen is enough for an average of 12 inseminations.

*R. R. Dykstra, D.V.M.*

# Failure to Establish Prenatal Ascarid Infection in Swine

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Experimental work reported here and on page 134 indicates that swine ascarids do not infect pigs in utero.

In a recent paper Kelley *et al.*[5] reported that pigs born from sows which had been treated and kept on ground previously uninhabited by swine showed ascarid lesions in the liver. In explaining the probable method of infection of the above pigs, the authors were of the opinion that the infection might have resulted from low egg production from residual worms in the treated mothers or possibly by wind blowing the eggs from nearby infected hog lots. In the above case, prenatal infection was not excluded, although as far as known from the literature, this form of infection in swine has not been recorded. Martin[5] is the only one who has reported failure to produce prenatal infection in swine experimentally.

Prenatal helminthiasis has been reported in pups with the dog ascarid, *Toxocara canis*,[1,2,6,8] the hookworm, *Ancylostoma caninum*,[2,8] and the blood fluke, *Schistosoma japonicum* (Yutoc[8]). Soliman (Lapage[4]) refers to rare records of the presence of larvae of both *Dictyocaulus filaria* of sheep and *D. viviparus* of cattle, in the fetus *in utero*. The following is a brief account of an experiment to determine the possibility of producing prenatal infection with *Ascaris suum* in swine. The observation was carried out for further confirmation of the report by Martin.[5]

## Experimental Procedure

In this study a 3-year-old sow, bred on October 6, 1959, was utilized. The sow had been raised on ascarid contaminated ground. No eggs, however, were found present in the feces of the animal at the beginning of the experiment. The eggs used to infect the sow were obtained from the uteri of living worms (*Ascaris suum*) and cultured at room temperature (25 C.) in 1% formaldehyde solution in shallow dishes. The eggs were used after 30 days, at which time they were found infective as determined by their ability to produce infection and lesions in lungs of young guinea pigs.

The infective ascarid eggs were fed to the sow by first mixing the eggs in a small amount of granular animal charcoal, and then mixing the charcoal in a handful of dry feed placed in a trough. This was carried out in the morning before the animal received its normal feeding. The 1st eggs were fed to the sow on December 17, 1960, which was 72 days after the animal was bred. Thereafter, the eggs were fed once a week for a total of 5 weeks. During the first 4 weeks the sow received about 200,000 eggs at each feeding, and on the 5th week, 500,000 eggs. On January 28, 1960, which was 2 weeks after the last experimental infection, the sow gave birth to 13 normal piglets.

Immediately after farrowing, 8 of the young pigs were isolated in a clean wooden box. Four of these were killed within 1 hour after birth and 2 each after 24 and 48 hours, respectively. At necropsy the lungs, liver and intestinal tract of each animal were isolated. The lungs and liver were ground up separately with a meat grinder and examined for larvae by the use of the Baermann apparatus having a metal screen 20-mesh to the inch. The small intestine was slit open with an enterotome in a beaker containing warm saline solution. This solution was then examined for ascarid larvae by the use of sedimentation technic.

## Results and Conclusions

The feeding of about 1,300,000 infective ascarid eggs to a sow over a period of 5 weeks and during the last third of its pregnancy produced no apparent ill effect on the animal nor on its offspring. No ascarid eggs were found in the feces of the sow up to 55 days after the last experimental infection. The failure of the worms to become established and mature in the intestinal tract might have been due to resistance acquired by the animal from previous ascarid infection.

After the above sow farrowed, no lesions or ascarid larvae were noted in the liver, lungs or intestinal tract of 8 of the newborn pigs examined at necropsy from 1 to 48 hours after birth.

The above findings confirm the earlier observation of Dr. H. M. Martin that prenatal infection with *Ascaris suum* is probably not of common occurrence in swine.

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# Evidence Against Intrauterine Infection by Swine Ascarids

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Recent investigators, Sprent,[1] Douglas,[2] have found that various members of the nematode family Ascarididae commonly utilize intrauterine migration as a means of gaining access to new hosts. Douglas[2] found that larvae of *Toxocara canis* lie dormant in the bitch until about the 40th day of pregnancy, when they again begin migrating and move into the fetus. Herlich and Porter[3] established *Neoascaris vitulorum*, the bovine ascarid, in 1 of 5 unborn calves. These findings have again motivated questioning whether *A. suum* is capable of intrauterine transmission.

In 1929, Martin[4] fed several million embryonated eggs to 4 bred sows and later examined their pigs for ascarids. All were negative. Van Der Wall[5] (1958) fed 600 to 12,000 infective eggs to 3 sows 21 to 10 days before parturition. Examination of 6 piglets immediately after farrowing and of the remaining 17 pigs 10 weeks later, failed to reveal any ascarids.

## Experimental Procedures

An additional attempt to demonstrate intrauterine transmission of A. *suum* was recently undertaken in this laboratory. A Yorkshire gilt was fed 300,000 embryonated eggs of *A. suum* 5 days of each week for the last 5 weeks of gestation. At term, the pigs were taken by hysterectomy and placed in isolation units. Two pigs were killed immediately for necropsy. The remaining 4 were maintained 21 days before being examined. During the last 2 of these 3 weeks, the pigs were fed a high roughage diet with water and no milk. The intestinal tract, internal organs and carcasses of all the pigs were thoroughly examined by trituration and digestion. No larvae were found.

This study supplies supporting evidence that *A. suum* is probably not generally transmitted transplacentally.

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# Anthelmintic Activity of Four Organic Phosphates in Cattle

These observations of the effectiveness of certain organic phosphates as anthelmintics were made on beef and dairy cattle. The compounds were administered as a spray, bolus, drench or feeding premix.

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The chief interest in the organic phosphates, or the so-called systemic insecticides, has been in their application for the control of several arthropod parasites of domestic animals. However, recent investigations[1-4, 6-13] indicate that this group of compounds may also be characterized by a broad spectrum of activity against the gastro-intestinal nematode parasites of ruminants.

The present observations on the anthelmintic activity were made in conjunction with field trials on the use of several of these compounds for cattle grub *(Hypoderma spp.)* control.[5]

## Methods

Nine experiments were completed using a total of 431 animals including both beef (Hereford and Angus) and dairy (Holstein-Friesian, Jersey, and Brown Swiss) types.

The compounds tested and the methods of application were as follows.

Ronnel (Trolene—Dow Chemical) (0,-0-dimethyl 0-2,4,5 trichlorophenyl phosphorothioate) was administered (1) in the bolus form at the recommended rate of 100 mg/kg. body weight and (2) in the form of a 10% premix in the ground grain ration. Two levels were fed, (1) seven 15 mg./kg. daily doses, and (2) three 35 mg./kg. daily doses; both of these delivered a total dose of 105 mg./kg. The technical grade of Ronnel (Korlan—Dow Chemical) was applied as a 0.75% spray in one experiment.

Dimethoate (Compound CL 12,880—American Cyanamid) (0,0-dimethyl S-$\alpha$-mercapto - N - methylacetamidodithiophosphate) was fed in the form of a 10% premix in the ground grain ration. Five 3 mg./kg. daily doses were planned for one experiment, but treatment was discontinued on the 4th day when several animals showed symptoms that appeared to be a toxic reaction to the drug.

0,0-diethyl 0-(3-chloro-4-methyl-7-coumarinyl) phosphorothioate (Co-Ral—Chemagro) was used as a 0.25% spray in one experiment.

4-tert-butyl-2-chloro-phenyl methyl methylphosphoromidate (Ruelene — Dow Chemical) was given at the rate of 44 mg./kg. as a drench in 2 experiments.

Pre- and post-treatment worm egg counts (EPG) on feces were made to evaluate the action against the nematode parasites.

Usually, the pre-treatment count was made on rectal fecal samples taken on the day of treatment just before its application, and the post-treatment count was made one week later. In Experiments 2 and 6, additional post-treatment counts were made at 1- or 2-week intervals for 1 and 8 months, respectively.

The worm infections were naturally acquired, and although only total egg counts are reported here the infections were mixed. Several species of trichostrongylids were represented, and on the basis of the egg characteristics they were predominantly of the Cooperia-Ostertagia-Trichostrongylus type. The Haemonchus-Oesophagostomum type was present in much smaller numbers, while Bunostomum, Nematodirus, Strongyloides, and Trichuris were present in very small numbers.

## Results

The groups, treatments, and egg count data for the nine experiments are summarized (Table 1).

The effect of ronnel treatment on body weight gain was measured in Experiments

**TABLE 1. Fecal Egg Count Data on Activity of Organic Phosphate Insecticides Against Internal Parasites of Cattle**

| Exp. No. | Animals Type | Animals No. | Treatment Compound | Treatment Rate | Treatment Method | Worm Eggs/Gm Feces Number Counted | Before* Range | Before* Average | After** Range | After** Average | % Change |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Hereford Steers | 177 | Dimethoate | 3 x 3 mg/kg[x] | Feed | 30 | 0-960 | 155 | 0-690 | 125 | -19 |
| | " " | 35 | Ronnel | 3 x 35 mg/kg | Feed | 17 | 10-460 | 195 | 0-170 | 40 | -79 |
| | " " | 10 | Untreated Controls | — | — | 10 | 10-480 | 220 | 30-360 | 165 | -25 |
| 2 | Dairy Calves | 6 | Ronnel | 3 x 35 mg/kg | Feed | 6 | 10-980 | 390 | 10-300 | 90 | -77 |
| | " " | 6 | Ronnel | 7 x 15 mg/kg | Feed | 6 | 20-1130 | 410 | 10-390 | 115 | -72 |
| | " " | 7 | Untreated Controls | — | — | 7 | 60-1330 | 470 | 0-1410 | 300 | -36 |
| 3 | Dairy Heifers | 16 | Ronnel | 100 mg/kg | Bolus | 16 | 0-550 | 125 | 0-80 | 25 | -80 |
| | " " | 14 | Ronnel | .75% | Spray | 14 | 0-480 | 135 | 0-760 | 110 | -19 |
| | " " | 14 | Untreated Controls | — | — | 14 | 0-730 | 165 | 0-710 | 185 | +12 |
| 4 | Angus Heifers | 20 | Ronnel | 100 mg/kg | Bolus | 10 | 10-70 | 44 | 0-10 | 3 | -93 |
| | " " | 20 | Untreated Controls | — | — | 10 | 10-100 | 42 | 0-80 | 31 | -26 |
| 5 | Angus Calves | 13 | Co-Ral | .25% | Spray | 6 | 0-60 | 25 | 0-70 | 23 | - 8 |
| | " " | 12 | Untreated Controls | — | — | 6 | 0-40 | 22 | 0-40 | 17 | -23 |
| 6 | Dairy Calves | 8 | Ronnel | 100 mg/kg | Bolus | 8 | 50-380 | 215 | 0-30 | 10 | -96 |
| | " " | 8 | Untreated Controls | — | — | 8 | 100-680 | 295 | 80-2510 | 650 | +120 |
| 7 | Dairy Calves | 5 | Ronnel | 100 mg/kg | Bolus | 5 | 10-210 | 84 | 0-10 | 4 | -95 |
| | " " | 5 | Untreated Controls | — | — | 5 | 0-60 | 34 | 0-60 | 26 | -24 |
| 8 | Dairy Calves | 6 | Ruelene | 44 mg/kg | Drench | 6 | 130-180 | 146 | 10-80 | 20 | -86 |
| | " " | 6 | Untreated Controls | — | — | 6 | 10-400 | 153 | 10-340 | 110 | -28 |
| 9 | Hereford Steers | 15 | Ruelene | 44 mg/kg | Drench | 9 | 0-770 | 218 | 0-50 | 7 | -97 |
| | " " | 20 | Phenothiazine | 10 gm/cwt | Drench | 13 | 0-1,110 | 210 | 0-70 | 24 | -89 |
| | " " | 8 | Untreated Controls | — | — | 7 | 40-320 | 167 | 0-340 | 146 | -13 |

* On day of treatment.
** Seven days of treatment.
x—Scheduled 5 day course, stopped on 4th day because of toxicity.

1, 2, and 6. These observations are recorded in Table 2. Gains during the 4-week interval after treatment were greater, but they were not significantly different from the untreated animals in either Experiment 2 or 6. This was of interest because the EPG's during this interval revealed that treatment with three 35 mg./kg. doses via the feed and the boluses at 100 mg./kg. markedly reduced EPG's for at least 2 weeks. Appreciable recovery of EPG was recorded on the 4th week. More transient reduction of EPG was associated with the seven 15 mg./kg. dose course of treatment in Experiment 2 because a moderate increase was recorded on the 2nd week. Extended observations for 2 to 6 months post-treatment failed to reveal statistically significant differences of weight gains between treated and control animals in Experiments 1 or 6. A gradual loss of worm infections was observed during this interval in Experiment 6 until relatively low counts were posted on the 20th week.

## Discussion

Although Ronnel and Ruelene were the only compounds exhibiting anthelmintic activity in the present observations, all of the compounds except Ruelene were tested and found to be effective against the grubs (Hypoderma) as recorded elsewhere[5].

Method of administration or application played a very significant role in determining anthelmintic action by the group of compounds, whereas this factor is relatively less restricted[5] with respect to determining antigrub activity. Ronnel *per os* as a bolus or premix in the grain ration produced marked reductions in fecal EPG, but application of the same compound as a spray had no such effect on the worms. Likewise Co-Ral spray did not reduce EPG, although this chemical has been critically tested[7] and shown by others[8] to be quite effective against most gastrointestinal nematode parasites of cattle when larger doses are administered as a drench. Generally higher concentrations of the drugs in the gastrointestinal tract would result from *per os* administration, and these observations suggest that anthelmintic action is probably dependent on this factor.

The observed effectiveness of the small repeated doses of ronnel in the feed is somewhat at variance with the foregoing; however, the added factor of cumulative effects must be considered with this method of treatment. Although feeding a series of small daily doses produced a marked reduction of EPG in the aggregate, considerable variation between animals was recorded. This points up the hazard of treating groups of animals by feeding an anthelmintic. Nonetheless, the average EPG reduction was significant, and the general feasibility of this method of administration emphasizes the desirability of further investigation of the feeding method of treatment. Recent reports[10,12] indicate that feeding Ruelene

**TABLE 2. Fecal Egg Count (EPG) and Body Weight (Pounds) Data on Ronnel (R) and Dimethoate (D) Treated Cattle**

| Exp. No. | Animals No. | Treatment Rate | Treatment Method | Pre-Treatment EPG | Pre-Treatment Wt. | Post-Treatment (Week) 1 EPG | 1 Wt. | 2 EPG | 2 Wt. | 4 EPG | 4 Wt. | 8 EPG | 8 Wt. | 20 EPG | 20 Wt. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 177 | D-3 x 3 mg/kg | Feed | 155 | 624 | 125 | — | — | — | — | — | — | — | — | 907** |
| 1 | 35 | R-3 x 35 mg/kg | Feed | 195 | 619 | 40 | — | — | — | — | — | — | — | — | 935* |
| | 10 | Untreated | — | 220 | 666 | 165 | — | — | — | — | — | — | — | — | 973* |
| | 6 | R-3 x 35 mg/kg | Feed | 390 | 393 | 90 | — | 30 | — | 180 | 450 | — | — | — | — |
| 2 | 6 | R-7 x 15 mg/kg | Feed | 410 | 361 | 115 | — | 230 | — | 100x | 436x | — | — | — | — |
| | 7 | Untreated | — | 470 | 399 | 300 | — | 275 | — | 215 | 445 | — | — | — | — |
| 6 | 8 | R 100 mg/kg | Bolus | 215 | 332 | 10 | — | 45 | — | 255 | 364 | 140 | 394 | 20 | 471 |
| | 8 | Untreated | — | 295 | 331 | 650 | — | 430 | — | 570 | 361 | 210 | 392 | 30 | 457 |

x = Average for five calves.
* = 21 weeks.
** = 23 weeks.

in small daily doses is effective against gastrointestinal parasites of sheep.

In this study, the effectiveness of treatment was evaluated by changes in fecal EPG. Post-treatment reduction of EPG could be due to removal of the worms and/or inhibition of egg production. Greater EPG reductions were associated with Trolene than seemingly could be expected from the critical tests data[6] on this compound in cattle and would suggest that additional suppressive effect on egg production was involved. On the other hand, tests with a related phosphate compound in sheep by other investigators[1] showed close agreement between changes in EPG and worm recovery data. Also, in 2 experiments of the present study, the series of EPG's at intervals after ronnel treatment showed that the reductions lasted between 2 and 4 weeks. Such a long interval also corresponds with the prepatent period of the dominant worm species involved and suggests that the observed reductions in EPG were in fact an index of the removal of the mature worms and not a transient post-treatment suppression of egg production.

Improved weight gain following organic phosphate therapy reported[11] in other studies has been ascribed to (1) destruction of grubs and (2) removal of nematode parasites. In the present observations, both of these actions followed ronnel therapy which was also associated with greater body weight gains for the treated animals than for the corresponding untreated control animals. However, both the gain increases and the animal numbers in the experiments were relatively small, and although a trend was evident the statistical analyses showed that the greater gains were not significant.

This investigation is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director of the Station and the Director of the Animal Disease and Parasite Research Division, U. S. Department of Agriculture. It is in cooperation with the Animal Disease and Parasite Research Division, Agricultural Research Service, U. S. Department of Agriculture, and it was conducted as a part of Southern Regional Project S-21, "Internal Parasites of Ruminants."

## Summary

The anthelmintic activity of 4 organic phosphate compounds, ronnel (Trolene and Korlan), Co-Ral, dimethoate, and Ruelene, was evaluated by pre- and post-treatment fecal egg counts. These observations were made in conjunction with field trials on grub *(Hypoderma spp.)* control and involved 431 beef and dairy animals. Except for Ruelene, which was not tested, the other compounds were variously applied and were effective against the grubs. However, only Ruelene as a drench and ronnel administered *per os* as a bolus or via the grain ration produced marked reductions in post-treatment egg counts. Ronnel's application as a spray showed no effect on worm egg counts. Likewise, Co-Ral spray and dimethoate *per os* in the grain ration did not reduce fecal egg counts after treatment.

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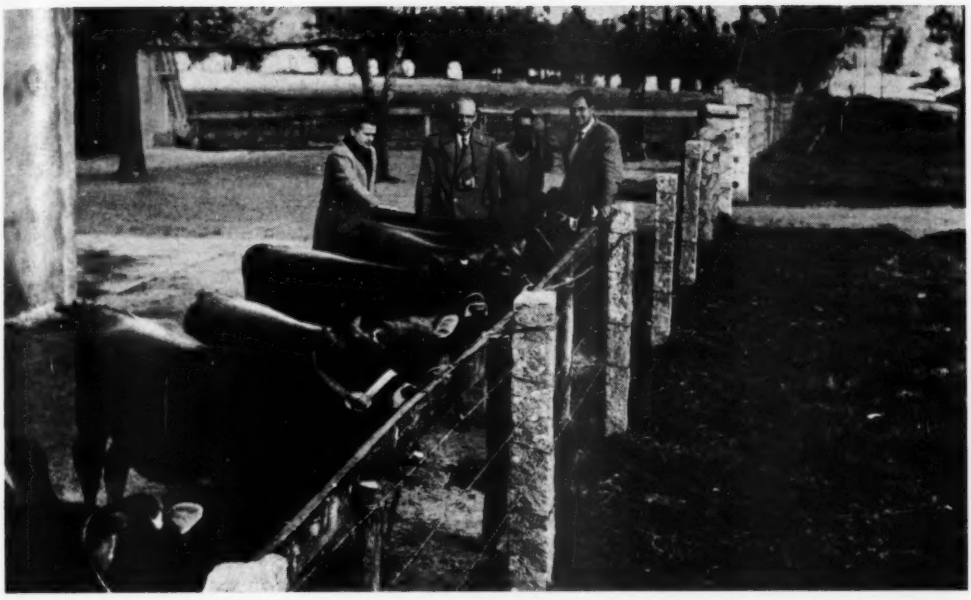

*Dr. Fincher (2nd from left) with his associates, Dr. Hans Merkt, a leader in the veterinary clinics at Porto Alegre, Brazil (on the far right); the herdsman of a fine Jersey herd at Pelotas and (on the far left) Mr. James Robinson working with the I.C.A. at Porto Alegre. The line-up of fine type Jersey bulls is proof that frozen semen shipped to this farm in Pelotas from the Wisconsin area was successfully used.*

# Report on South American Tour of Dr. Fincher, Cornell Professor

M.G. Fincher

M. G. Fincher was granted a leave of absence from Cornell University to travel to South America to give lectures and demonstrations on bovine mastitis, sterility in cattle and horses, and to hold group discussions on various other problems of interest to agriculturists, veterinary faculties, students and official government veterinarians. The 6-week trip, sponsored by the International Educational Exchange Service, United States Department of State, Washington, D. C., began on June 23, 1960.

The cities in which the veterinary profession was contacted included Lima, Peru; Santiago, Chile (an unscheduled stop); Montevideo and Pando, Uruguay; Porto

Alegre (2 short visits), Pelotas, Curitiba, Sao Paulo, Campinas, Belo Horizonte, Rio de Janeiro (Kilometer 47), all in Brazil. Veterinary colleges, when located in these places, were visited. Horse and cattle breeding farms and experiment stations were also visited.

The veterinarians and other people interested in livestock production were most appreciative and cooperative. Judging by the excellent publicity in several city newspapers and in agricultural journals, a visit by a veterinarian from a faculty of a veterinary college in the United States was a big event and one that the agriculturists and veterinarians would like to have repeated.

*A view of a clinic at the veterinary college at Porto Alegre with Dr. Hans Merkt, formerly of Hannover, Germany (on right of Dr. Fincher) and a group of other faculty members and government veterinarians observing and discussing one of several sterile cattle, presented for examination.*

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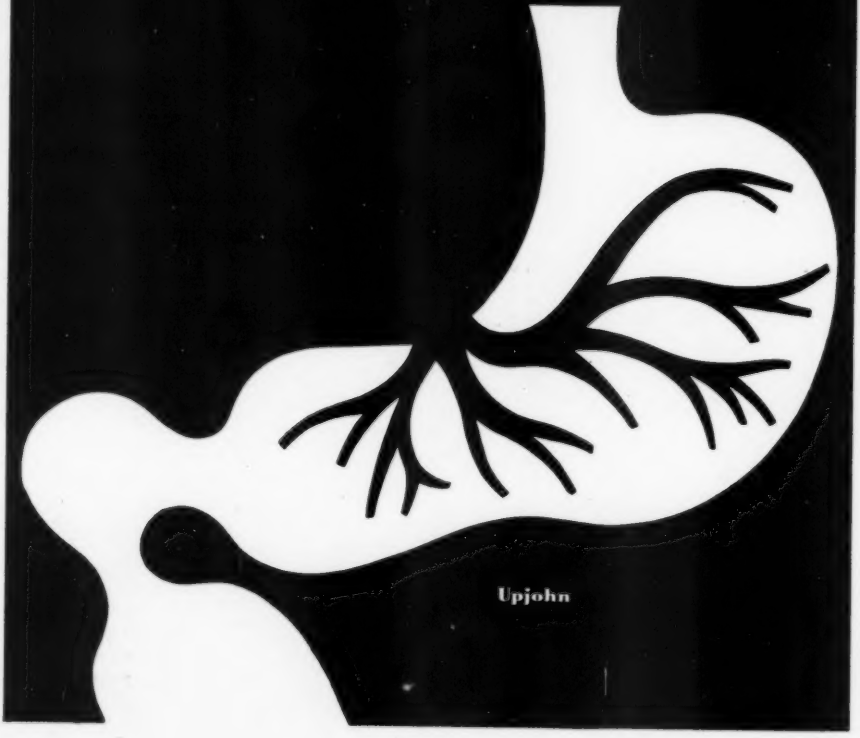

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